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ENCYCLOPÆDIA MEDICA

Foot. *See* Ankle-Joint, Tarsus, Operations.

Foot and Mouth Disease.

The term foot and mouth disease is applied to an eruptive febrile disorder of a markedly contagious nature which affects ungulates, ruminants more especially. The eruption is vesicular, and after the vesicle has burst a very hyperæmic and sensitive corium is exposed. The disease is known under a variety of names, which express the opinions that have been held from time to time of its nature; such names are epizootic aphtha, aphthous fever, eczema epizootien, eczema contagiosa, the vesicular disease, murrain epidemic, etc. It is probable that the disease is an exotic, because, according to the *Annual Report of the Board of Agriculture* for 1898, there is no evidence that the disease ever existed in this country prior to 1839, though the Germans say that foot and mouth disease was rife in England in the middle of the eighteenth century. Whether such is the case or not, we know from Continental authorities the disease was known abroad during last century, and that in the main it was well described by contemporary authorities. Since 1839 there have been several serious outbreaks of the disease in Great Britain, and during the first two years following 1870, when the Board of Agriculture first obtained returns of the diseases affecting stock, it was shown that 1,149,124 animals had been attacked with foot and mouth disease. In 1894 the disease was eradicated, and the chief veterinary officer estimates that it claimed for its victims from 1839 to its final extinction between ten and eleven millions of head of stock. Since 1894 there have been isolated outbreaks of the disease, the last during February of the present year in Norfolk and Bedfordshire. The disease is always existent upon the Continent, in the Low Countries especially, and it is very probable that the occasional outbreaks we have to contend with may have their origin traced to the commerce with these countries. Fortunately the fatality is not great among animals other than those at the teat; the percentage of fatal cases varies from 1 to 5 per cent; in young animals, however, where the alimentary viscera may show lesions, the percentage may mount as high as 50 per cent or more.

Transmission of the disease to the human subject is of fairly frequent occurrence, especially on the Continent, though less often in this country. The disease may attack only one or two individuals, or may occur in epidemic form. In the course of an epidemic many persons in one locality have been affected, and cases may be observed to terminate fatally. The number of cases recorded in the human subject probably falls far short of representing the actual figures, on account of the difficulty in many instances of making a correct diagnosis. The first case was recorded in 1695. Since then many epidemics have occurred in different countries, and more especially between 1862 and 1869 in this country, though even in 1884 over two hundred persons suffered from the disease in Dover. In Germany, a large number of cases are still recorded each year.

ETIOLOGY.—The virus is contained in the saliva, the milk, urine, fæces, and skin secretions, and retains its vitality for months, even for a year. Since the secretions and dejections are infective, it has been observed that the disease spreads along the great trade routes from the east of Europe to the west, and along the railway trunk lines, healthy animals being contaminated by the cattle-trucks, stubbles, pastures, and byres that have sheltered the diseased, and by manure, litter, fodder that have been soiled by the infective secretions. The disease is also spread by the attendants upon the animals, milkers, cattle and sheep dealers, and others brought in contact with healthy stock, after having handled those suffering from foot and mouth disease. It is known that birds suffer from the disease, and these may assist in spreading the disease.

The *bacteria* which have been found in connection with the disease possess merely an historic interest, and none of them represent the specific causal agent, which has not yet been discovered. Numerous bacteria have been found mainly in the vesicles: "Micrococcus aphtharum" (Rivolta and Nosotti), streptococci (Klein), "Streptococcus involutus" (Kurth), and pleomorphic bacteria (Stutzer and Hartleb, van Niessen). The Streptococcus involutus appears to be a normal inhabitant of the mouth of cattle (Sanfelice). A bacillus was obtained by Bussenius and Siegel, chiefly from affected animals, but also from the human subject, in fatal cases. The bacillus somewhat resembles the members of the Coli group, and Siegel has lately admitted that it is not the specific agent. Furtuna and Starcovici, Sauer, and Babes and Proca have also described bacteria in relation to the disease. Bodies resembling protozoa were found by Schottelius, Behla, Piana and Fiorenti, and Jungers, but these bodies are probably not true protozoa, and no etiological significance is to be attached to them. The Report published in 1897-98 of the German Commission which investigated the disease, shows that as a rule no micro-organisms can be detected in fresh vesicles, but if the vesicles are several days old, various bacteria have usually gained entrance into their interior.

The specific virus is chiefly contained in the lymph of the vesicles. The blood serum does not seem to be infective after the local signs of the disease have appeared. Lymph which has been mixed with water and then passed through an unglazed porcelain filter is found to be infective. Hence the filtered lymph must either contain a toxine, which is poisonous in such a degree of dilution ($\frac{1}{750000000}$) as is hardly credible, or else the supposed micro-organism is so small that it can pass through the pores of the filter. The latter is the more probable explanation, for it has been shown that the disease can be transmitted from one animal to another in succession by means of such filtered lymph. Another point in favour of the virus being a corpuscular substance, is the fact that diluted lymph which has been repeatedly passed through a Kitasato filter loses its virulence (Loeffler and Frosch). Lymph retains its infective power for weeks or months under ordinary conditions, but loses it if exposed to a temperature of 70° C. for half an hour, or to 60° C. for one hour.

Experimental Infection.—Calves and cattle are the animals most susceptible to infection, pigs being less so. The most reliable method of infecting an animal is by the introduction of fresh lymph into the blood-stream. By this method $\frac{1}{5000}$ c.c. of lymph will cause the disease, but $\frac{1}{50000}$ c.c. fails to cause infection. Infection usually occurs after the introduction of slaver from affected animals into the mouths of healthy ones, but with greater certainty if the mucous membrane be previously scarified. The subsequent disease after such an inoculation is as a rule milder than after natural infection. Infection can take place through the stomach, but it is doubtful whether it occurs after subcutaneous inoculation of lymph.

Immunity may or may not be acquired after natural infection. Thus, an animal after recovering from an attack may be immune for months or even years, or on the other hand an animal may suffer repeatedly from the disease. In other cases, again, immunity has been acquired *in utero.* The Commission already referred to investigated a number of important facts in regard to the production of *artificial immunity.* It was found that no immunity was conferred by intravenous inoculation with fresh lymph in doses so small that the disease was not produced, or by subcutaneous inoculation with lymph if the disease were not thereby caused. Nor was immunity acquired with certainty after the use of lymph rendered inactive by heat, nor after inoculation with the blood of animals which had recovered from the disease, and which had themselves been found to be immune. This latter statement confirms the results previously obtained by Schütz and David and Zernecke. Siegel, however, stated that he had obtained a protective serum from animals which presented the specific vesicular eruption, and healthy animals inoculated with this serum were said to resist without any special reaction subsequent inoculation with infective lymph. Hecker also lays claim to having obtained a protective serum from the blood of immunised cattle. The German Commission further found that immunity may be produced by the cutaneous injection of a mixture of foot and mouth disease lymph and vaccine lymph. Calves and pigs are said to acquire immunity by the intravenous injection of a mixture of foot and mouth disease lymph and "immune blood," and the injection of these two substances separately has a similar if less powerful immunising action. In the case of adult animals, one is advised to leave the lymph and the serum in contact with one another for some time before injecting the mixture. Diluted lymph, which has become non-virulent by being repeatedly passed through a Kitasato filter, is likewise said to protect animals without causing the disease.

It is, however, somewhat doubtful whether these statements concerning artificial immunity are really reliable, and at present we are unable to say that there is any method whereby true artificial immunity may be produced. Inoculations are doubtless to some extent protective, but any method aiming at the production of a high standard of immunity tends to cause the disease itself. This fact is well exemplified by the results following inoculation with the "Seraphthin" of Loeffler and Frosch. Seraphthin has neither afforded protection nor rendered milder any subsequent attack, and in some instances it has even appeared to cause an outbreak of the disease against which it was supposed to be protective.

Animals attacked.—Though the domestic ruminants (ox, sheep, goat) are most commonly attacked, the pig is very susceptible, and the horse, dog, cat, and fowls are not exempt. It has also been observed in the camel, llama, giraffe, deer, antelope, buffalo, bison, etc. The disease may be transmitted to man, either by inoculation or by the consumption of milk from diseased cows, or by butter and cheese made from contaminated milk. Cream especially is virulent.

Symptoms.—The symptoms will be described under the two heads of constitutional and local.

Constitutional Symptoms.—The incubative period of the disease varies between twenty-four hours and seven days. Three or four days is common. During invasion the animals show an elevation of temperature, but this need not necessarily be high; in many outbreaks a rise of 2° F. alone has been noticed. The animal isolates itself, stands with the back arched, and has rigors. Saliva may escape from the mouth, and the patient may move stiffly. There may or not be slight abdominal pain, and the same may be said of cough. The respirations are not increased in number usually, though in some cases in the later stages the respiratory movements may be much increased in frequency. The pulse-rate is slightly quickened. If the animal is milking or nursing, the amount of milk furnished by the gland falls off and the quality of the milk is changed; it is of a yellowish white colour and not unlike colostrum in appearance.

Local Symptoms.—The local symptoms in the bovine affect the mouth and the feet. Lesions, however, may be observed upon the skin, mammary gland, vulva, or prepuce of the male. In the sheep, mouth lesions are not common, and in the pig the lesions also are generally confined to the feet, though the snout may also show the eruption.

In the Bovine Mouth.—Prior to the appearance of the vesicles the mucous membrane of the gums and of the lips may appear to be injected, and the amount of saliva present is greater than usual. On the third or fourth day a crop of vesicles put in appearance. These are at first small—no larger than the head of a pin; but they rapidly increase in size by continuing to develop, or by several uniting. The vesicles are found upon the gums, mucous surfaces of the lips, tongue, dorsum, and sides. The vesicles of the dorsum are larger than those upon the sides of that organ, the hard palate, including the pad covering the toothless premaxillæ. As we have mentioned, the vesicles may become confluent, and individual vesicles may attain the size of a half-crown or more. The fluid contained in the vesicles is at first clear like to water, but later becomes opaque. If the vesicle be ruptured the corium is exposed and found to be intensely hyperæmic. The lesion only involves the superficial parts of the corium, not intruding any great depth into that structure. The epithelium around the wound forms a rounded edge, and soon extends over the denuded corium, restoring the lost covering. Pus is not commonly found upon the exposed tissues, which are constantly flushed by the enormous amount of saliva produced. The epithelium over large areas may be lost; extensive patches from the sides and tip of the tongue, and from the anterior third of the mucous membrane of the hard palate, may be removed. Save in very malignant cases the excoriations are healed in a week or ten days, but a cicatrix is left behind; the papillæ upon the affected parts are not restored. The lesions may extend into the pharynx, and give rise to difficulty in swallowing, or into the trachea and bronchi, and produce cough.

Feet.—The lesions are observed upon the skin in front, just above the hoof (termed by veterinarians the coronet), in the interdigital space, and upon the heels behind. The rudimentary digits may also exhibit the lesion. Vesicles are formed which, on bursting, expose an intensely inflamed corium. Pus frequently is found upon the inflamed surfaces, but usually a scab speedily covers the wound, and healing proceeds rapidly. Sometimes, however, a necrosis sets in and extends deeply, producing an arthritis, a necrosis of the bones, and often, especially in pigs, a loss of hoof. Death results frequently from septicæmia. The foot lesion, which attacks one, two, or even all the feet, induces lameness, and when the animal is standing in a stall the foot is frequently shaken as if to rid it of an offending object. A smacking of the lips, between which saliva is dribbling away, and shaking of a limb in the manner indicated, and lameness when the animal is moved, are almost diagnostic. The presence of the eruption, its character, and several animals manifesting the same symptoms and lesions, make diagnosis certain. The lesions, when observed upon the skin and mucous membrane of the vulva, upon the mammary gland, or upon the prepuce and skin, are similar to those observed upon the feet and have a similar course.

In Sheep.—The lesions in the sheep are generally confined to the feet, upon the coronet, and most often towards the heels. The animals affected go lame, and linger behind their companions when travelled. The disease may in these animals be taken for "foot-rot," a common disease which is not contagious, but in "foot-rot," which is primarily a disease of the foot, the lesions commence at the toe, and work upwards, whilst in foot and mouth disease the lesions, if the horn-forming structures are involved, extend from above downwards.

In the pig the disease affects the feet most commonly, and in this animal loss of the hoof is not rare. Occasionally lesions are observed upon the snout, and the vesicles may reach a great size.

In the horse the disease is confined to the mouth, but the constitutional and local symptoms in the mouth are similar to those observed in the bovine. Variola and a contagious form of stomatitis may have been taken for foot and mouth disease, but there is at least one authentic case—the subject had licked a cow suffering from the disease.

The disease has also been noticed in the dog and cat. It is also known to attack fowls, vesicles being developed in the mouth and pharynx, upon the conjunctiva, comb, and the membrane between the toes.

In young of all species the disease is serious, and in addition to the local external lesions gastro-intestinal disturbance is indicated, and not infrequently proves fatal. Indeed, the greatest fatality is to be expected among the young at the teat, and milk fed from cows suffering from the disease.

The disease is probably transmitted to man by milk taken from infected cows, or by butter and cheese prepared from such milk, or by direct inoculation from animals suffering from the disease.

The following examples illustrate the methods of transmission: Three veterinarians intentionally drank milk from affected cows; a girl chewed a piece of wood smeared with saliva of a sick animal; again, a wounded hand or finger came in contact with infective buccal secretion; and lastly, an injury was received from the teeth of a sick animal. The symptoms at first are malaise, headache and fever, diarrhœa, and in some cases a rigor and vertigo. About three to five days later, the temperature falls to about normal, and one sees a catarrhal inflammation of the mucous membrane of the mouth and pharynx, accompanied in about one-third of the cases by the formation of small vesicles. These may also appear on the lips, fingers, and toes. The stomatitis in some cases is so severe that small ulcers are formed on the mucous membrane, especially of the gums or the tongue. The urine in most cases remains normal. The duration of the disease is about four weeks. In exceptional cases one finds a skin eruption resembling measles, or vesicles on the skin of the trunk.

The diagnosis in the human subject is often difficult, especially in sporadic cases. The disease may be mistaken for aphthous stomatitis, herpes labialis or pemphigus, and the atypical forms may resemble measles or even typhoid fever if the intestinal symptoms be very pronounced.

Post-mortem Appearances.—Few adult animals die from foot and mouth disease without complications: a septicæmia or a pyæmia may result from secondary infection. Lesions have been noticed, however, in the liver, spleen, and kidneys, changes perhaps induced by the poison of the fever. Ulcerations have been found in the larynx and pharynx, fatty degeneration of the myocardium and pneumonia.

In young animals gastritis and enteritis are not uncommon.

Treatment.—Treatment is simple and resolves itself into (*a*) hygienic, (*b*) dietetic, (*c*) local. The disease, being highly contagious, should be treated as such. The affected should be isolated, and a cordon drawn around the isolation station, and no living thing be allowed to cross the barrier save after rigid disinfection. All litter, refuse, dressings, etc. that have been in contact should be burned, and special suits should be provided for attendants that can be soaked when necessary with a disinfecting solution. The walls and floor of the house in which the diseased have been confined should be flushed down; if necessary scraped, and then washed with solution that will destroy the microbe. Under somewhat similar conditions a one per cent solution of sulphuric acid in water has answered well; crude carbolic acid, sulphate of copper, chloride of lime are useful and cheap. As to the diet, the animals will require cooked nutritious food, soft and sloppy, which in swallowing will produce as little discomfort as possible. Hard food will lacerate the mucous membrane, retard the healing of wounds in the mouth, and may aggravate gastro-intestinal lesions. Local treatment consists in the application of mild astringents and disinfectants to the mouth and to the feet. Mouth lesions are best dressed with borax and glycerine applied by brush; a dilute solution of chloride of zinc in water may be used for application to the feet. If many animals are affected, such as a flock of sheep or a herd of swine, these may be driven through a shallow trough, sunk a few inches into the ground, and containing in it an antiseptic solution. All that is required is that an area, about four yards long by three yards wide, be sunk about six inches below the general level of a paved yard. If the depression be made water-tight by cement, a shallow bath of antiseptic fluid is provided through which the diseased animals may be gently driven.

As to the flesh of foot and mouth patients it is innocuous and marketable, save when profound changes have occurred due to fever. The heads of cattle should be condemned and the feet of all. If the heads of sheep and pigs show lesions, they also should be destroyed. Hides and fleeces should be disinfected: they have undoubtedly in many outbreaks been the vehicles by which the disease has spread.

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Fourth Nerve. *See* OCULAR MUSCLES.

Fractures.

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It is impossible within the compass of this article to do more than bare justice to such an important surgical subject as the fracture of bones. Space, however, can be found to fill in the outline and to draw attention to the advances which improvements in physical methods have rendered possible in diagnosis, and to the changes which Listerian surgery has introduced into their treatment.

Causes.—The causes of fractures are either predisposing or determining. The *predisposing causes* are those abnormal conditions which lead to the atrophy of bone or to alterations in its consistency. Chief amongst them are rickets, syphilis, tubercle, and scurvy; new growths, either primary and sarcomatous, or secondary and carcinomatous, especially when they grow within the bone and weaken it by expansion; hydatids, and such physiological causes as old age, pregnancy, usually about the seventh month, and that interference with the blood-supply to the bone which is often associated with long-continued disuse. Some affections of the central nervous system are also predisposing causes of fracture, the best known being locomotor ataxy, progressive muscular atrophy, disseminated sclerosis, paraplegia, general paralysis, and the congenital defects of the central nervous system associated with hydrocephalus. There is also a constitutional brittleness of bone (osteopsathyrosis or fragilitas ossium) about which we know but little, except that it is sometimes hereditary, and is transmitted strictly in the male or female line.

The *determining cause* of a fracture is always injury, the violence acting either directly upon the bone, as when the ribs are broken by a kick from a horse, or indirectly, when they yield to pressure applied simultaneously to the sternum and the backbone, as often happens in a crowd. The amount of force required to break a bone varies very greatly, and the result sometimes appears to be out of all proportion to the cause, as in cases where a slight slip produces a severe compound fracture, comminuting the bone and lacerating the tissues, whilst in other cases the application of a much greater force may only cause a simple fracture. Such a variation seems to depend in part upon the direction in which the force is applied to the bone. A cylindrical bone resists to perfection a pull in its long axis or a weight applied in its diameter, but it offers very little resistance to a twist and still less to flexion, for there is no doubt that muscular action is able to break even so stout a bone as the humerus.

Indirect violence causes as many fractures as when the force is applied directly to the bone, but its results are very capricious. Thus a fall upon the hand may lead to a Colles' fracture at the wrist, to a fracture of both bones of the forearm, the ulna in the lower third, and the radius at a higher level; to a backward dislocation of the elbow; to a fracture of the humerus in its lower third, or the arm may escape entirely, the clavicle being broken obliquely at the junction of the middle and outer third. A fall upon the feet in like manner may cause a variety of fractures more or less distant from the legs, until it culminates in a fracture of the base of the skull.

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[1] These exclude fractures in the immediate neighbourhood of joints, for which see "Ankle-Joint," "Elbow-Joint," etc.

Congenital fractures occur from time to time, many of course produced during birth, but others—the truly intra-uterine—have taken place during gestation, for they may be found to have repaired themselves before delivery. Some of the intra-uterine fractures are undoubtedly the result of injury to the mother's abdomen, as in intra-uterine fractures of the leg, femur, and clavicle; others again are associated with embryonic malformation, whilst yet others are spontaneous in the sense that no adequate cause can be assigned for their occurrence.

THE CLASSIFICATION of fractures has undergone recently a very remarkable change. It was all-important for the older surgeons to discriminate between simple fractures, where the skin remained unbroken, and compound fractures, where the ends of the broken bone were exposed either directly or indirectly to the air. A simple fracture healed with them as it does now: a compound fracture always meant prolonged suppuration, and was too often the cause of death to the patient. Aseptic surgery has changed all this, and we now make a fracture compound with as little hesitation as we incise tissues elsewhere. Primary compound fractures, however, are still of grave prognosis, especially when they involve the larger joints, for it is impossible in many cases to render them surgically sterile. Our main classification, therefore, has become anatomical, and a fracture is grouped, first according to its position, and afterwards as to whether it is complete or incomplete. The complete fractures are further subdivided into single or multiple; simple or comminuted; oblique, transverse, spiral, or longitudinal; impacted or unimpacted; involving the joint or wholly extra-articular, for upon all these points depends the prognosis.

An incomplete fracture is either bent, curved, or indented. It is usually seen in children, the bent or curved variety in the tibia, radius, or ulna of the rickety, and indented fractures in the frontal or parietal bones after falls or the application of midwifery forceps. A greenstick fracture is another form of incomplete fracture, in which the bone is bent so that its convex surface is broken without any necessary laceration of the concave surface. It is often seen in the clavicles and in the bones of the forearm of children, and its name is derived from the resemblance which it shows to the behaviour of a green twig which has been forcibly bent. Except in the clavicle all greenstick fractures should be straightened before they are put into splints. This is especially necessary in the case of the radius and ulna where great impairment of pronation may result when the bone is allowed to heal in a bent position. In some cases a bone may be broken and its ends may be comminuted without rupture of the periosteum, a condition seen in children suffering from that form of scurvy known as "Barlow's disease." Fissured fractures, such as occur in the skull, the ilium, and the lower jaw, are also instances of incomplete fractures. The difficulties connected with incomplete fractures are rather those of diagnosis than of prognosis or treatment.

TRAUMATIC SEPARATION OF THE EPIPHYSES is only now beginning to receive the attention which the importance of the subject merits. Fractures and separated epiphyses were formerly considered identical injuries and were treated in the same manner, but surgeons now recognise that the two forms of injury are distinct, that they are attended by separate signs and sequelæ, and that they require different treatment. Traumatic separation of the epiphyses is most common between the ages of eleven and eighteen years, for in young children the epiphyses are so cartilaginous and elastic as to escape injury, whilst later in life they become fused with the shaft of the bone. The separation of the epiphysis is either complete or incomplete. When it is complete the line of separation extends through the epiphyseal line in its whole extent, or it involves a part of the shaft of the bone. Partial detachment or juxta-epiphyseal strain is apt to be overlooked or treated merely as a sprain.

It is often difficult to make an accurate diagnosis when the displacement is slight, but in well-marked cases unusual mobility at the seat of an epiphysis, local pain, with swelling and ecchymosis, afford a clue to the nature of the injury when the age of the patient is taken into account. If crepitus can be obtained it differs from that occurring in fracture because it is "muffled," that is to say, it lacks the crispness of bone moving upon bone, for it is caused by cartilage rubbing against the uneven end of the diaphysis. The delicate tissues of children lend themselves especially to the use of the X-rays, either by screen or plate, in the elucidation of these injuries.

The prognosis depends partly upon the nature of the injury and partly upon the treatment. Simple separations in healthy persons heal as readily and with less deformity than simple fractures, but in unhealthy persons abscess, periostitis, and tubercular disease are not uncommon. The especial dangers to be feared are paralysis and gangrene due to pressure of the displaced epiphysis; the more remote dangers are permanent deformity, impaired movement in the neighbouring joints, and either partial or complete arrest of growth in the limb. Infective osteomyelitis is sequel of compound separation of an epiphysis, though it may also occur in the simple forms.

The treatment of a separated epiphysis consists in immediate replacement in the compound as well as the simple forms of injury. When the displacement is considerable an anæsthetic must be administered, and reduction should be brought about as methodically as in a case of dislocation. The injured portion of the limb is then encased in some form of moulded splint, and the circulation through the part is carefully watched for a day or two. Massage is applied earlier than in fractures, and in a simple case may be commenced on the tenth day.

Every complete fracture is associated with a certain amount of displacement of the two ends of the broken bone, the amount depending upon the position of the injury and the condition of the ends of the bone. It is either sliding or angular, simple or twisted, in unimpacted fractures, and it is part of the art of surgery to render the displacement as slight as possible, and to keep the ends of the bone in the most accurate apposition until the injured part is able to repair itself.

DIAGNOSIS.—The diagnosis of fractures has greatly improved since the use of the Röntgen rays has enabled the surgeon to view the fragments on the screen and to obtain skiagraphs of the broken bones, because many injuries which were formerly recognised with the greatest difficulty are now rendered perfectly clear. In large towns, in public institutions, and in private practice, it is advisable to skiagraph every case of fracture a few days after the bone has been set, in order to verify the accuracy of the position. The negative can be obtained without moving the limb and in ordinary daylight, but it is necessary to use a wooden or plaster splint. The diagnosis of a fracture depends upon signs which can be verified by the surgeon, and symptoms which require the descriptive powers of the patient for their elucidation. The diagnosis of a fracture is often easy, but there are many cases in which it can only be made with the very greatest difficulty, and there are some in which the nature of the injury remains for ever undetermined, or is ascertained so late that the surgeon is held blameworthy.

METHODS OF EXAMINATION.—A routine method of examination should therefore be adopted in every case in which there is a suspicion of fracture, and although the symptoms are less trustworthy guides than the signs, they should be determined first by questioning the patient, who will thus become accustomed to the surgeon's examination. Pain and loss of power in the limb are the two chief symptoms of fracture of a long bone, although both may be fallacious. Pain is an important symptom if it can be shown to attain a maximum over a given spot directly after an injury, and if the same point of maximum pain is indicated when the bone is fixed above and pressure is made upon the shaft at some distance below the seat of injury. But the sudden onset of localised pain is a characteristic feature of some forms of acute inflammation of bone. On the other hand, absence of pain in a case of undoubted fracture raises a suspicion of chronic alcoholism (when a surgeon is on his guard for the appearance of delirium tremens or traumatic delirium), or it points to locomotor ataxy or other disease of the central nervous system. The loss of power over the limb is often a well-marked symptom of fracture, but it is absent in cases of impacted fracture, or when only one of a pair of bones is broken in the arm or leg. A simple contusion, however, especially over the hip, often causes great loss of function though the impairment is only temporary.

The signs of fracture are much more important than the symptoms. They should be elicited methodically, taking care to give the patient the least possible pain. The examination should be made at the earliest opportunity before the swelling has obscured the landmarks, and the least painful part of the examination should be undertaken first. In every case the injured part should be exposed completely, that the surgeon may examine it thoroughly, and it should be compared carefully with the corresponding part upon the opposite side. The examination should be continued until the surgeon has decided upon the diagnosis, for diagnosis in fractures is the key to successful treatment. In every case, too, care must be taken not to overlook other injuries when a fracture has been detected, especially in cases near a joint, where a dislocation may complicate the fracture. And even though a fracture is simple when it is first seen, the ends of the bone and the skin should be examined to ascertain whether it is likely to become compound.

Every fracture is associated with some deformity of the broken bone, although the deformity may be masked by the thickness of the overlying tissues, as in the hip, or by the support obtained from neighbouring bones, as in the ribs, the ulna, the radius, and the fibula. In fissured fractures the deformity is so slight that it may pass unnoticed.

Most fractures are associated with some alteration in the length of the injured bones, which become shortened, but the alteration may be so slight as to fall within the limits of error or of natural development. Every fracture of a long bone, therefore, must be compared carefully with the sound side by means of a tape measure, taking care to measure from similar bony points on the two sides.

Abnormal mobility in the shaft of a bone is a certain sign of fracture, but it is wanting in greenstick, curved, and fissured fractures as well as in the great group of impacted fractures.

Crepitus, or the sensation produced when the two ends of a recently broken bone rub against each other, is diagnostic of a fracture; but it may be mistaken by the inexperienced for the creaking of a tendon moving in an inflamed sheath, as in tenosynovitis; for the rustling of melon-seed bodies in a bursa or ganglion; for the slight stickiness which accompanies effusion into joints; for collections of blood containing blood-clots; and for the crackling of emphysema. There is no crepitus when the ends of the bone are widely separated, as in fractured patella, when there is impaction, or when a mass of tissue intervenes between the two fragments. Although crepitus, felt by an experienced person, is a certain sign of fracture, it is always undesirable to obtain it by rubbing the ends of the bone together. The aggregate of the other signs is usually sufficient to determine the nature of the injury, and the production of crepitus is very painful to the patient; besides, it may do harm to the surrounding tissues and even to the ends of the bones themselves.

An extensive ecchymosis, first seen some days after the injury, and the appearance at a still later date of the lump due to callus, are valuable aids to diagnosis in some very obscure cases, as in fractures about the hip and shoulder, and in greenstick fractures.

Process of Repair.—The pathology of the repair of fractures is difficult, because the details have not yet been completely investigated. The method differs somewhat in aseptic and in septic fractures. Aseptic or simple fractures are repaired by callus, a material produced by the fibrous tissues in the immediate neighbourhood of bone, and therefore derived from the bone itself, from periosteum, and from the surrounding connective tissues. The injury which produces the fracture tears all the soft parts so that the blood is poured out in considerable quantity both from the periosteum and from the vascular medulla. The blood remains fluid for six or eight days, by which time the periosteum near the fracture is swollen, and its fibres are dissociated by exudation and the proliferation of cells in its deeper layers. This forms the first or hæmorrhagic period in the repair of a fracture; it is of very short duration in such vascular bones as those of the face, which heal almost by first intention, and it is proportionately prolonged where severe injury has given rise to much laceration, or where disease, as in scurvy and hæmophilia, has led to a great effusion of blood. The second, embryonic or cartilaginous, stage is that in which inflammatory tissue is produced in abundance on every side by the periosteum, the Haversian canals, and the medulla. In adults the inflammatory cells are abundant and the cartilage cells are few, but in animals and in children the cartilage cells may be very numerous. The blood is absorbed and the embryonic tissue or blastema becomes converted into fibrous tissue, the organised callus. The portion of callus lying farthest away from the bone becomes organised more rapidly than that which lies between the broken ends and within the medullary canal. New bony tissue is then formed beneath the periosteum and within the medullary canal by the ordinary process of intra-cartilaginous or intra-membranous ossification, and this constitutes the third stage in the repair—that of provisional callus—whose formation and consolidation lasts on an average from fifteen to forty days.

The provisional callus is at first porous, but the final process of repair is one of modelling, which lasts for months or years. The modelling process is a combination of a rarefying osteitis at the periphery, combined with sclerosing osteitis at its centre, and it continues until the callus is converted into normal bone, and until all sharp edges, spicules, and fragments of the original bone have been rounded off. It must not be thought, however, that these stages exist independently of each other or have well-defined limits. They are all processes in the general train of repair, and so pass insensibly into each other.

Repair in a septic or compound fracture takes place in a manner similar to that just described, but the process is masked, hampered, and modified by the suppuration. The callus is less uniformly deposited, granulations are formed in abundance, and the bony fragments are joined ultimately by the union of granulations, which anastomose one with another, and rapidly become converted into bone.

Many pitfalls surround the repair of fractures. The process may be completed so quickly that the bone is firmly united in a bad position before the surgeon has set it, or even has realised that he is dealing with a fracture. This is most likely to happen in a broken nose or in a greenstick fracture. Repair may be unduly delayed on the other hand, or it may never take place at all, as is most frequent in the humerus, in the tibia, and in the femur, where it is difficult to keep the two fragments motionless. In other cases so much callus may be produced as to lead to serious difficulties by the pressure which it exercises upon the nerves or blood-vessels in the neighbourhood.

THE PROGNOSIS of a primary fracture involves two separate considerations, the danger to life and the danger to the limb. The danger to life depends in part upon the general condition of the patient, his age, and his freedom from renal and bronchial troubles; and in part upon the character of the individual fracture, which may lead to injury of the brain, lungs, or pelvic viscera. The prognosis in regard to the utility of the limb after a fracture must always be guarded, even in the most simple forms of injury, because, although the result depends to a great extent upon the vigilance of the surgeon and his attention to detail, there is an element of uncertainty which renders it most unwise to be too sanguine either as to the duration of treatment or the usefulness of the limb. Extensive series of examinations of fracture by means of the Röntgen rays have shown that perfect union is rare, and that oblique fractures generally have some overlapping of the broken ends, whilst in deep-seated bones there is often much bony deformity which it is impossible to recognise by the ordinary diagnostic methods. The prognosis becomes more grave, therefore, when the bone is much comminuted, when the line of fracture is very oblique, when it is close to a joint, and when the large vessels and nerves of the limb are injured.

It will be the duty of the surgeon in some cases to recommend amputation of the limb or excision of a joint in some cases of fracture. The operation is either primary or secondary. A primary amputation is required when the limb is hopelessly shattered, as in railway smashes, and in the extensive comminuted fractures caused by explosions, or the passage over a limb of such heavy and slowly-moving objects as drays and tram-cars. The duty of the surgeon is quite plain in these accidents, but there exist a great group of doubtful cases which often exercise the highest art of the surgeon to decide whether he should amputate at once or should wait. On the one hand he may sacrifice a limb unnecessarily, on the other he may cause the death of his patient by delay. Young and healthy adults may recover after excessive laceration of the soft parts with extensive comminution and denudation of the bone, after implication of a large joint, after the main vessel has been ligatured, and the main nerve sutured. In children and boys I have a great distrust of compound separations of the epiphyses, especially when they occur at the shoulder and knee, for osteomyelitis readily occurs, and the patient dies even when amputation has been performed. Old people, I think, should undergo amputation in doubtful cases, whilst children and adults should receive the benefit of the doubt. Secondary amputations are often more hazardous than primary operations, because the condition of the patient is less satisfactory. A secondary amputation is required in gangrene, where there is septic absorption associated with osteomyelitis or extensive suppuration, and as a last resource in cases of non-union or union in such a bad position as to render the limb useless.

TREATMENT.—Success in the treatment of a fracture depends upon the maintenance of the broken ends of the bone in the best apposition possible until they are soundly healed, taking care that the surrounding tissues are neither crippled by disuse nor are involved unduly in the accompanying inflammatory changes. The objects to be attained by treatment in every fracture of a long bone are firm, bony union, good position, and a useful limb. The treatment of fractures resolves itself, therefore, into reduction with subsequent fixation.

Reduction is the manipulation employed to bring the broken ends of the bone into their natural relation to each other. It may be necessary, useful, or harmful; easy, difficult, or impossible. Reduction without any delay is necessary in a depressed or punctured fracture in the skull, since cerebral symptoms may be produced; in a broken nose, because the nasal bones unite so rapidly that the deformity very soon becomes permanent; and when the sharpness of the fragments threatens to convert a simple into a compound fracture. Reduction is useful in nearly every simple fracture of a long bone, but it may be positively harmful in some cases of impacted fracture, as in those connected with intra-capsular fractures of the hip and shoulder, or in a fracture of the outer table of the skull. Reduction is easy in most fractures, but impaction, muscular spasm, or the interposition of the soft tissues, may render it difficult or even impossible to bring the two ends of the broken bone into good apposition; although early reduction is desirable in most fractures. No attempt should be made, therefore, to reduce a fracture until the surgeon is ready to "put it up" permanently, and until everything is ready the injured part should be kept at rest by a temporary splint. A bandage and sling may be sufficient for the arm, but in the case of the leg the limb must be supported by improvised splints, by securing it to a pillow with a bandage placed above and below the seat of fracture, by sand-bags, or by the help of an assistant to restrain the awkward and involuntary movements which may so easily convert a simple into a compound fracture, or may even drive a sharp fragment of bone through a large artery or vein.

Such care is especially needful when the patient has to be moved from the place of accident to his house or to the hospital, since helpers are more often willing than handy.

Reduction is effected by extension and counter-extension, the force being applied whenever it is possible by assistants, that the surgeon may have his hands free to manipulate the injured part and to apply the splint as soon as he has satisfied himself that the position of the broken ends is sufficiently satisfactory. There are many cases in which mere force will avail nothing, and the surgeon must resort to a variety of manœuvres before he can reduce the fracture, and allusion will be made to this in dealing with the fractures of individual bones. Anæsthesia is often a valuable aid when reduction is difficult, but the surgeon who has to treat such a case in private must bear in mind that the patient's struggles whilst he is becoming unconscious, or unskilled efforts to restrain them, may lead to serious injury at the seat of fracture.

The broken limb must be fixed securely as soon as it has been "set," for it is characteristic of fractures that the deformity is reproduced as readily as it is reduced. It is usually impossible to obtain perfect apposition, for the deformity is produced by the whole of the soft parts surrounding the bone, and not by the action of the muscles alone as was formerly thought; still the deformity can often be diminished by a skilful surgeon.

Fixation is of the greatest service in cases of fracture. It lulls the pain, allays or prevents inflammation, and favours repair with the least possible amount of callus. But the method of fixation has been carried to excess, for it has been used to the exclusion of all other methods of treatment. It has, too, the disadvantage of causing atrophy of the bones, muscles, skin, and connective tissue, whilst the joints and tendons may become hampered from prolonged disuse. It is not surprising, therefore, that a very justifiable reaction against the employment of fixation has taken place during the last few years. The reaction has been carried to an extreme by some surgeons, but there can be no doubt that the early release of a limb from splints, passive movements, skilfully applied shampooing, and the wiring of certain fractures, has been attended with much better results than the older practice of complete fixation carried out rigorously for many weeks. Every case must be treated on its merits, and not on a principle of routine. Success depends very largely upon the vigilance of the surgeon, and upon the attention which he gives to details apparently trivial and minute.

Splints and plaster of Paris bandages or cases form at present the most usual and easiest methods of securing physiological rest for a fracture.

The variety of splints is infinite, and it passes the ingenuity of man to number them. It may be taken for granted that the simpler the splint the more useful it will be, and that a surgeon with the help of a carpenter and a blacksmith should be able to manufacture all that he is likely to want in his everyday practice. Wooden, plaster of Paris, and poro-plastic splints are better than metal, for they do not interfere with the passage of the Röntgen rays; and whenever it is possible a skiagraph of the limb should be taken after the bones have been duly set and secured in a splint. Wooden splints are made of white pine, the width selected being always a little greater than that of the limb, so that injurious pressure may be avoided. The splint must be well and firmly padded with tow, the fibres being drawn roughly parallel to each other and sewn into a cover of old linen. The skin must be well washed with soap and water, gently dried and dusted over with oxide of zinc or starch powder, and all bony prominences should be protected by padding round them, not over them, with absorbent wool. In no case must skin be allowed to touch skin if the two surfaces are to be kept at rest, and a little wadding or a layer of lint must be placed between them. A bandage must never be put round the limb beneath a splint, and as a rule no bandage should be applied over the seat of fracture. The splint is attached to the limb by strips of plaster, which must encircle the limb spirally and never circularly. It is a cardinal rule that if a splint is uncomfortable it must be adjusted and readjusted until the patient no longer complains, though at first it is always irksome. The limb must be carefully watched after the application of a splint to see whether it swells. Œdema is a sign that there is some interference with the circulation, and a defect in the application of a splint is the most common cause of swelling after a fracture.

Massage combined with fixation appears to give better results in many cases of fracture than the use of splints alone. Some surgeons trust to massage and rest, discarding the use of splints, but this I believe to be dangerous, and I have obtained most satisfactory cures by following the method adopted by Mr. W. H. Bennett, who thus describes the technique (*The Lancet*, vol. i. 1898, p. 359): "The treatment is very simple, and is easily acquired by any person of ordinary intelligence possessing a light hand and a fair sense of touch, gentleness being the keynote to successful manipulations. The method comprises three stages: (i.) Gentle rubbing in an upward direction over the fracture with a view to soothing the patient, the relief of muscular spasm, and the rapid absorption of extravasated blood, etc.; (ii.) Passive movements of the joints above and below the fracture (thus effecting 'internal massage'), by which all matting of the soft parts at the seat of fracture and about the joints is prevented; (iii.) The development of wasted muscles by the ordinary massage processes. The details will be best understood by describing an ordinary straightforward case of fracture of both bones of the leg three or four inches above the ankle, in which there is little or no difficulty in keeping the bones in fair position. Reduction of any displacement of the fragments having been accomplished, the limb is placed upon a back splint reaching above the knee, with a footpiece to which the foot is fixed by a bandage in the ordinary way, care being taken to include no more of the leg above the ankle than is absolute necessary; a second bandage or piece of wadding fixes the limb to the splint just below or at the knee. As much as possible of the area of the fracture should be left exposed. Rubbing by a gentle, smoothing movement upwards from the ankle is now made by the flat of the hand, grasping as much of the circumference of the limb as is possible. However tender the parts may seem at first, no pain will be caused, but on the contrary a soothing effect is rapidly produced. Ten minutes of this rubbing is sufficient at the first application. If at the end of this time the patient is fairly comfortable, the toes are taken altogether between the operator's thumb and fingers, and very gently extended upon the metatarsal bones two or three times. At the end of the 'sitting' side splints or sandbags are used in addition to the back splint for the better steadying of the fracture. This proceeding is repeated daily, or oftener if practicable, for a period of four to seven days, the time occupied by each massage being gradually increased to twenty minutes or more—the side splints being removed before the commencement of each rubbing and afterwards replaced. At the end of this time, if the fracture is in good condition and the fragments show no sign of altering their position, the bandages are removed from the foot and ankle, leaving the limb exposed and lying upon the splint. The smooth rubbing already described is now applied over the foot, ankle, and leg for about ten minutes, and then, without removing the leg from the splint, the operator gently flexes the ankle two or three times or more on the leg with one hand whilst he steadies the fracture with the other, the bandages being afterwards replaced as before. This is repeated daily for three or four days, after which the limb at each sitting is gently lifted off the splint on to a flat pillow; the rubbing is now more thoroughly done, and the passive movements of the ankle more freely carried out, the fracture being still, of course, supported with one hand of the operator; at the end of each sitting passive movement of the knee is now added. The passive movement of the ankle must be commenced very gently, as some slight pain may be caused by 'the internal massage' resulting from the working of the tendons and muscles in immediate relation with the fracture itself. At the end of another week the union is generally sufficiently firm to allow of all the manipulations of ordinary massage, and the patient may be encouraged to move the ankle spontaneously as freely as possible, the fracture being fixed with some form of short splint. The complete massage should be continued until the union has firmly consolidated; the period necessarily varies in different cases, but in a simple, uncomplicated case of fracture of both bones of the leg a month is the approximate time. For the first fortnight the patient is better confined to bed; after that he may lie on the sofa, and generally be allowed to get about on crutches, in which case a moulded poro-plastic or leather splint, made so that it is easily removable for the massage sittings, may be desirable." In the early part of the treatment of fracture by massage it is essential to success that the broken ends of the bone should be kept absolutely at rest, and unless the surgeon shampoos the part himself, or entrusts it to an experienced person in whom he has implicit confidence, it is better not to adopt the method at all, but to leave the massage until the ends have united. Massage is not necessarily contra-indicated in compound fractures. Especial care must be taken in such cases to prevent the infection of the wound. Shampooing is unsuitable when the skin is extensively injured, when there has been so great an effusion of blood just beneath it as to form a large hæmatoma, and where from the seat of the injury it is difficult to keep the fragments in apposition, as in fractures in the upper third of the humerus and the lower third of the femur.

Ambulatory.—The judicious application of various forms of splint, plaster case, or plaster bandage, is often of service in allowing a patient with a broken leg to go about his business before the fracture is firmly united, and this is particularly useful in a fractured fibula, or when a portion of the internal malleolus of the tibia has been torn off. Such ambulatory treatment of a fracture requires that the patient should be kept strictly under observation, as the dependent position of the limb or the imprudence of the patient may easily cause a disaster. The foot and leg must be separated from the plaster of Paris case by a padding of cotton wool at least an inch thick, and this padding must be further increased at the heel, so that in walking the patient puts his heel to the ground before the toes on the injured side.

Wiring.—Even simple factures are now often treated by cutting down upon the ends of the bone, wiring, pegging, or screwing them together, and afterwards closing the wound. This treatment is most frequently used in transverse fractures of the patella, where a tedious and incomplete convalescence extending over many months is thus replaced by a rapid recovery within a few weeks. Every patient with a broken knee-cap must not be treated in this manner, nor every fracture. A healthy person in the prime of life with a fracture across the middle of the patella gives the best results, but even then the surgeon must be certain of his own ability to carry his methods to an aseptic issue, for an operation which fails often costs the patient his limb and may seriously endanger his life. But the operation of wiring is by no means confined to fractures of the patella. It is often serviceable in very oblique ununited and badly-united fractures, though so far as my experience goes it is most unsatisfactory in the treatment of ununited fractures occurring in children. Simple or aseptic fractures may be wired when injuries to the nerves or blood-vessels require an incision at the seat of injury, whilst many cases of compound fractures likely to become septic can be wired at the time the wound is enlarged to disinfect the injured parts.

SEQUELÆ.—The consequences or sequelæ of fractures are many. *Stiffness* is the commonest, due either to the existence of adhesions between the muscular and tendinous sheaths and the injured bone, to degeneration and contraction of the muscles, or to the formation of adhesions in a joint which is not perfectly healthy, or which has been injured at the time of the fracture. Massage or the hot air treatment is often of the greatest service in these cases. If the hot air treatment be adopted the limb should be well covered with flannel, and the moisture should be reduced to a minimum by occasionally ventilating the apparatus. Each sitting, after the first, should last an hour, with the temperature beginning at 300° F. and rising rapidly to 380°-400° F. The impairment of movement may be caused by the overlapping of the united ends of the broken bone, by venous troubles associated with injury to the veins, or to thrombosis starting in the medullary veins. These, however, are comparatively trivial consequences of fracture, and appropriate treatment will usually cure them in a longer or shorter time.

Delayed union, fibrous union, and non-union are more serious, but fortunately much less common sequelæ. An ununited fracture is sometimes the result of such constitutional causes as scurvy and chronic nephritis, which weaken the patient, but it is much more often the result of causes acting locally. Any mechanical impediment to the apposition of the ends of the bone may lead to non-union, and of these the intervention of a piece of torn muscle, part of the aponeurosis, or the synovial membrane of a joint, are the most common. But want of rest is the most frequent cause of an united fracture, for delayed union or non-union is least often seen in the practice of those who are most careful to keep the ends of the bone quiet and in apposition. Slight rotatory movements of the ends of the bone upon each other do not seem to influence the process of repair, but hinge movements may entirely prevent it. For this reason care must be exercised in treating children's fractures by plaster of Paris bandages, especially in cases of broken leg. Their limbs soon shrink from disuse, and a plaster case which fitted admirably at first may become too large in the course of a week, free movement being thus allowed between the ends of the bone. The result is most disastrous, the bones atrophy, all attempts to cause union fail, and the limb becomes so useless as to require amputation. The prognosis in adults is better, for delayed union may often be remedied by putting up the fracture more securely and for a longer period, whilst non-union may be treated successfully by wiring or otherwise keeping the two ends of the bone in accurate apposition.

COMPLICATIONS.—The complications of fracture are very numerous, and it is only possible here to allude to some of the most common and important. Amongst the general complications are shock, exhaustion, collapse from hæmorrhage, delirium, œdema of the lungs, and bed-sores. Amongst the local complications of fracture are the various conditions which tend to convert a simple into a compound fracture, and foremost amongst these are sharp ends and excessive bruising of the skin, with so much extravasation of blood into the subcutaneous tissues as leads to sloughing. A large *extravasation of blood* may therefore be laid open with advantage, the blood being washed out and the skin afterwards brought together with interrupted sutures. Other local complications which require the attention of the surgeon are coexisting dislocations and injuries to nerves, arteries, and veins at the seat of fracture. Osteomyelitis, necrosis, pyæmia, and infective or spreading gangrene are more especially complications of compound fractures. *Gangrene* may occur from a variety of causes, constitutional as well as local. It may be infective from the presence of a micro-organism, in which case it is usually fatal, or it may be associated with diabetes or other disease leading to general debility. On the other hand, mortification after a fracture may be purely mechanical in its origin. It is then associated with severe bruising, with acute inflammation in badly-nourished tissues, or to pressure of bandages unduly exercised. Care must be taken, however, in these cases to eliminate other causes before the surgeon commits himself to an opinion that the gangrene has been produced by the pressure of splints or bandages applied by another person. I well remember the case of a man who had broken his ulna, in which the house surgeon was blamed for allowing such a catastrophe to occur. Subsequent dissection showed that the radial artery had been tied years before on account of a punctured wound, and that the ulnar had been pressed upon by the broken end of the bone until the blood-supply had become insufficient for the needs of the part.

Some *clotting of the blood* in the torn veins and medullary sinuses takes place after every fracture, and if the thrombosis is extensive it is an important factor in the œdema which is so troublesome a complication of many fractures. Portions of the clot may be carried away and may lodge as emboli in the smaller branches of the pulmonary and cerebral vessels. These emboli have been observed at various times after the fracture, but they are most common between the twenty-second and thirty-seventh day after the injury. When the medulla of the bone has been extensively crushed, or when it has been softened by inflammation, portions of liquefied fat may be carried to the lungs, brain, and spinal cord, where they block the capillaries and form "fat emboli." These *fat emboli* are not to be mistaken for the post-mortem clots containing fat globules which are sometimes seen in the bodies of persons who have had diabetes. The true fat emboli are generally found where there has been much crushing of the bone associated with large openings in the veins, and so with extensive extravasations of blood. They are produced earlier than the ordinary embolus from thrombosis, so that the symptoms are generally observed within a few days of the fracture. Their presence is marked by transient attacks of dyspnœa, irregular action of the heart, slight hæmoptysis, and Cheyne-Stokes breathing. Collapse with marked pallor of the skin and mucous membrane soon ensues, with spasms of various kinds, or bilateral paralyses and a diminution of reflex irritability. The chest is free from dulness or râles. The temperature varies so much in the different cases recorded that it is useless as a guide. No secondary abscesses are formed, but fat has been detected occasionally in the urine. Packard thinks that such a condition should be treated by the intravenous injection of ether and by the administration of diffusible stimulants.

Muscular spasms occurring in the injured limb are often a very painful complication of fracture. They appear about the sixth or seventh day after the injury, and are especially troublesome at night. They are most often caused by some slight error in setting or fixing the broken bones, and they can often be cured by taking the injured limb off the splint and carefully readjusting it. If they persist in spite of local treatment a suspicion of chronic alcoholism, or of an unstable nervous system, may cross the mind of the surgeon. In very exceptional cases muscular spasms herald the coming of tetanus.

CONSTITUTIONAL SYMPTOMS.—First in importance amongst the constitutional consequences of fractures is that condition, often associated with old age, in which confinement to bed leads to the group of symptoms known as "hypostatic congestion." Inability to sleep, a little cough slight wandering of the mind, associated with a progressive rise of temperature, show that the patient's heart is too weak to perform its functions, that the tone of the vaso-motor system is impaired, and that the lungs, having lost much of their elasticity, are little more than fibrous bags. Confinement to bed in such cases leads to œdema of the lungs and a pneumonic condition which soon ends in death. The patient must therefore be propped up in bed, or placed upon a couch, to secure the most favourable condition for his pulmonary circulation, but the surgeon need not abandon hope of obtaining good bony union, for he will make use of some of the many forms of plaster of Paris appliance.

Delirium is not an uncommon accompaniment of fracture. Traumatic delirium coming on within a few days of the injury is seen both in children and in old people. It is sometimes met with in overwrought people who have traded too much upon their reserve of nerve force. The delirium is of a low, muttering variety without any rise of temperature, and it has but little to distinguish it, except the cause, from delirium tremens. The prognosis is bad in old people, good in children and the middle aged, if it shows signs of yielding to the ordinary remedies of rest, sedatives, and diet. Delirium tremens is still far too common after fractures in every class of society. The onset is often very rapid. The delirium is active, and may be without any rise of temperature, though there is nearly always a temporary albuminuria. The patient shows a remarkable insensibility to pain, which is most disastrous to his perfect recovery, for he often succeeds in making a simple fracture compound, and he always prevents any accurate apposition of the ends of the bones in spite of every precaution the surgeon can adopt. The principles of treatment are to evacuate the contents of the stomach and large intestine, to restore the appetite, to procure sleep, and to prevent syncope. There is a third form of delirium associated with septic absorption, and associated, therefore, with a rise of temperature. This delirium is worse at night than in the daytime, and it ends when the suppuration is fully established.

A broken bone often remains a source of trouble after it has healed. The callus may be so exuberant as to lead to pressure or impairment of movement; it may be painful, especially in rheumatic, gouty, syphilitic, and agueish persons, or the pain may be due to direct inclusion of a nerve. Callus has been known occasionally to disappear altogether, so that a fracture which seemed to be firmly united has become ununited. It is not very uncommon for a sarcoma to appear at the seat of a fracture; bony and cartilaginous tumours have also been observed.

FRACTURE OF THE CLAVICLE.—A broken collar-bone is perhaps the most common fracture in childhood and youth. The frequency with which it occurs diminishes after the age of twenty, and it is a rare accident in persons over fifty years of age. Fracture of the shaft of the bone is more often the result of indirect than of direct violence, so that it is most commonly produced by falls upon the shoulder, upon the elbow, or upon the outstretched hand. Direct violence causes a fracture at the part struck, and though there are well-authenticated instances of a healthy collar-bone being broken by muscular violence, this accident is most often seen in bones weakened by such chronic inflammatory conditions as scurvy or syphilitic periostitis. The fracture is rarely compound, multiple fracture is not very uncommon, and both bones may be broken.

Immediately after the accident, in the case of simple fracture of one clavicle, the patient nurses his elbow with the opposite hand, and inclines his neck towards the injured side to relax the trapezius and sterno-mastoid muscles. This attitude is not absolutely characteristic of a broken collar-bone, for it is assumed in fractures of the scapula and in other injuries of the shoulder, but it serves to direct attention to the clavicle. The pain varies greatly in amount. It is not severe in a transverse fracture with only slight displacement, but in a very oblique fracture with much displacement the pain is great, and is caused partly by the sternal fragment pressing against the skin, and partly by the pressure of the outer end upon the large nerve trunks which pass below the clavicle.

There is usually no difficulty in making a diagnosis of the oblique fracture at the junction of the outer with the middle third, which is the ordinary result of indirect violence. The affected shoulder is narrower and more sloping than its fellow, whilst the bone lies so superficially that it is easy to feel any inequality. The displacement is considerable, the outer fragment being drawn downwards by the weight of the arm, forwards and inwards by the pectoral muscles, and it is at the same time pulled behind the inner fragment, and somewhat rotated upon its axis by the action of the serratus magnus and pectoralis minor muscles, which pull the scapula forwards and inwards, whilst the rhomboidei draw up its lower angle. The sternal end of the clavicle practically maintains its natural position. Crepitus is easily felt when the shoulder is pulled outwards or when the acromion is depressed.

Forcible transverse compression of the upper part of the thorax occasionally breaks both collar-bones at the same time. The patient is then rendered particularly helpless as the use of both arms is impaired. Dyspnœa is a frequent and important symptom. Simultaneous fracture of both clavicles is not necessary or even usually fatal, but it is often complicated by other injuries. The patient must be kept in bed and in the position best calculated to relieve his difficulty in breathing, and it should be remembered that there is a marked tendency for the fragments to override.

The *prognosis* is good in uncomplicated fractures, but the patient should be warned that there will always be some deformity at the seat of fracture, and that it may be a long time before he regains the power of swinging his arm round his head. In exceptional cases there will be shortening, and in a few cases I have seen non-union, though the arm has been useful. A broken collar-bone may be only a part of more severe injuries, and in such cases the surgeon should satisfy himself that there is no paralysis due to pressure upon the underlying nerves, that the axillary artery and vein are intact, and that the pleura and lung remain uninjured. The formation of exuberant callus is sometimes a very troublesome complication of fractured collar-bone, and it may be necessary to perform an extensive surgical operation to remove it. The fracture—especially in children—is often incomplete, and it is then very likely to be overlooked until the formation of callus makes the injury conspicuous, when the medical man is blamed because he has not applied a bandage. These incomplete or "greenstick" fractures are often seen in babies who have fallen out of bed, or who have been dropped by careless nurses, so that the history of the accident may be designedly misleading. For this reason I have long taught my pupils that when a baby or young child is brought with the complaint that it has pain and impaired movement at the point of the shoulder, it should always be treated as though the clavicle were fractured, even though no other signs of such an injury can be detected.

Treatment.—Any method which keeps the shoulders well back and the injured arm at rest is sufficient treatment for a broken collar-bone. Sayre's method (Figs. 1 and 2) is the one usually adopted in this country. Two pieces of stout strapping are taken measuring three inches in width by five feet in length. One piece of strapping is loosely stitched with the sticky side outwards round the injured arm opposite the insertion of the deltoid. An assistant is directed to draw the shoulder downwards and backwards to stretch the clavicular fibres of the pectoralis major, and to reduce the displacement of the outer fragment of the clavicle. As soon as this is done the surgeon carries the strapping backwards round the body so that the sticky side adheres to the skin (Fig. 1). He then stitches the end, which passes between the side and the arm to the encircling band in the middle of the back. The elbow of the patient is then brought forwards so that the hand lies flat upon the opposite shoulder, and the second piece of strapping (Fig. 2) is carried obliquely across the sound shoulder in such a manner that the point of the elbow on the injured side is received into a slit made in the strapping, whilst the hand and arm are also covered. A flannelette bandage is applied over the strapping to keep everything in place. The position of the arm is an important factor in correcting the displacement, and the more the outer fragment is displaced downwards, the more necessary is it to keep the upper arm vertical (Fig. 3); if the displacement is inwards the upper arm must be placed well across the chest (Fig. 4), whilst if the forward displacement is the most marked, the upper arm must be placed as horizontal as possible (Fig. 5).

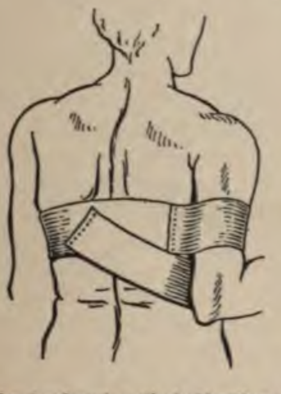


FIG. 1.—Sayre's method of treating a broken collar-bone; first step.

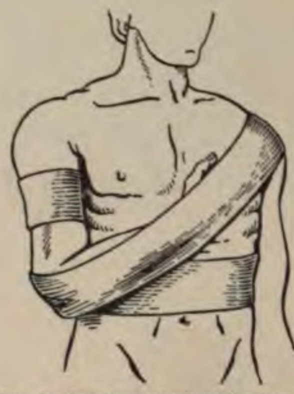


FIG. 2.—Sayre's method of treating a fractured collar-bone; second step.

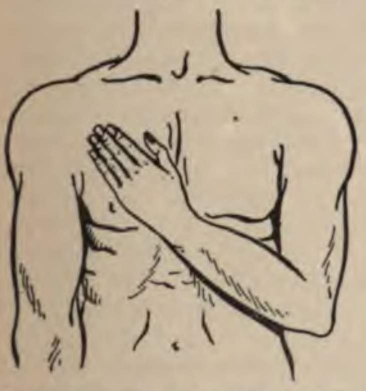


FIG. 3.—Diagram to illustrate the vertical position of the upper arm necessary to obtain the best results when there is a downward displacement of the outer end of a broken collar-bone. (Chiene and Dobie.)

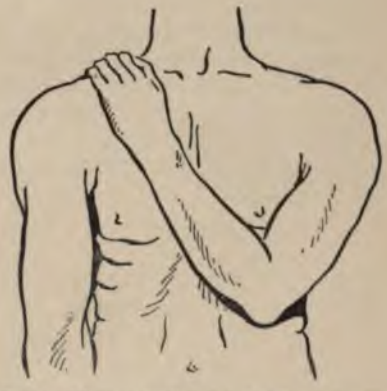


FIG. 4.—Diagram to illustrate that the upper arm must be placed well across the chest when there is much inward displacement of the outer end of a broken collar-bone. (Chiene and Dobie.)

Sayre's method is unsuited to men who have much hair upon their chests, and it is badly borne by ladies with delicate skins. It is often convenient, therefore, to employ one of the two following methods:—(1) Lay the tail of a bandage on the hand (Fig. 6), carry it down the back of the forearm, round the elbow, up the upper arm to the tip of the acromion (it is too far forward in the diagram), across the back to the opposite axilla, across the forearm, round the elbow again, up the upper arm, then round below the deltoid and across the back to the opposite shoulder, repeating these turns as often as may be necessary. Last of all, fix the arm to the side by a few circular turns of the bandage. A layer of boracic lint or cotton wool dusted over with zinc oxide is to be placed between the arm and the trunk to prevent skin touching skin. (2) The "first-aid" method is often serviceable (Fig. 7). A square yard of linen is cut diagonally to make two triangular bandages, and a small pad of cotton wool dusted over with starch powder or oxide of zinc is placed in the axilla. One of the triangular bandages is then placed beneath the arm of the injured side in such a manner that the point of the triangle lies behind and on a level with the elbow, the arm being bent and the tips of the fingers resting upon the opposite arm. The upper end of the long side of the triangular bandage lies upon the sound shoulder, and the lower end is brought upwards over the injured arm and beneath the injured armpit until it can be tied to the upper end on the sound shoulder. The second bandage is then folded "shawl-wise" to make a narrow bandage, which is carried round the body to keep the injured arm at rest. For children it is often sufficient to put a sling round the wrist and to dress the patient with the injured arm inside the clothes. In the case of a lady, where it is important to obtain union with the least possible deformity, the patient must be content to remain in bed, flat upon her back, and with only a very narrow pillow under her head, the arm being fixed in a sling or by a plaster of Paris dressing. But even then the tilting of the scapula directs the outer end of the clavicle downwards, and somewhat rotates it upon its own axis, so that it is almost impossible to keep the ends of an oblique fracture of the shaft of the clavicle in perfect apposition. A fortnight in children and three weeks in adults is generally sufficient to ensure good bony union in simple cases of broken collar-bone, but this time is insufficient to allow of perfect consolidation, and a man who has to do manual labour should not be allowed to resume work for five weeks.

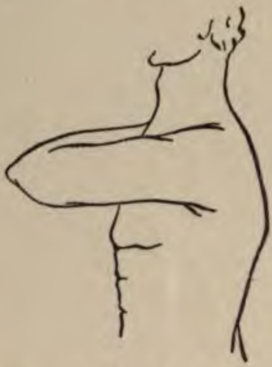


FIG. 5.—Diagram to show that the upper arm must be placed as horizontal as possible when there is much forward displacement of the outer end of a broken collar-bone. (Chiene and Dobie.)

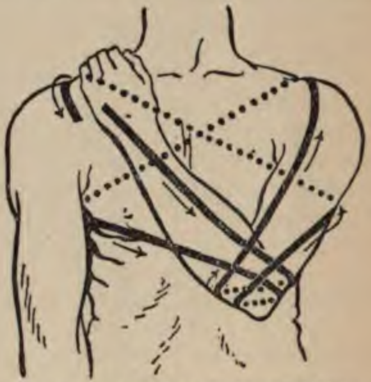


FIG. 6.—Bandage for a broken collar-bone when no strapping is used.

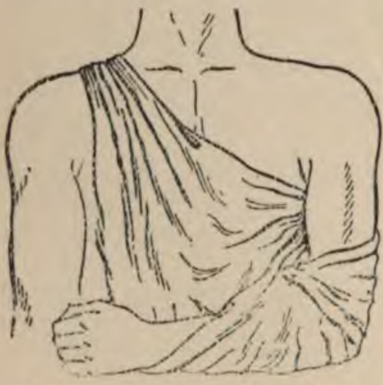


FIG. 7.—The "first aid" bandage for a broken collar-bone. (Manual for the R.A.M.C.)

Multiple fractures, double fractures, and complicated fractures of the clavicle, inasmuch as they are always produced by greater violence, require the patient to be kept in bed for their satisfactory treatment.

Fracture of the sternal end of the clavicle is so rare an accident that only thirty-one cases are recorded—twelve due to muscular action, sixteen to violence, and three in which the cause was not stated. It is probable that in every case the accident has been rather a separation of the epiphysis than a true fracture of the sternal end of the bone. The treatment is the same as for a fracture of the shaft of the clavicle.

Fracture of the outer end of the clavicle is considered under the article "Shoulder Joint."

THE SCAPULA.—Fractures of the scapula are not very common, but they take place at any part of the bone, because they are nearly always caused by direct violence. Fractures of the coracoid process and of the glenoid cavity are described in the article "Shoulder Joint." There remain to be considered fractures of the body of the scapula and of the acromion.

The *body of the scapula* may be broken by direct violence at the middle and at the upper or lower angle, or the fracture may be star-shaped. The exact nature of the injury may be very difficult to determine, as it is often associated with serious injuries elsewhere which prevent any thorough examination of the patient. There is often much swelling due to extravasation of blood; it is not always easy to obtain crepitus, and the effusion of blood in the connective tissue beneath the muscles causes a sensation which may be mistaken for crepitus; the scapula lies beneath a mass of muscle or adipose tissue in muscular and fat patients. When the body of the bone is broken, however, it is possible to discover a point where the local pain is greatest, and there is generally impaired movement of the arm when it is thrust forwards or horizontally. There may be considerable displacement when the upper or lower angle of the bone is broken, due to the action of the levator anguli scapulæ and of the serratus magnus muscles respectively.

The *prognosis* is good except in cases complicated by other injuries. The arm should be supported in a sling for four or five weeks, and adhesive plaster should be applied to the injured side of the back in the manner best calculated to keep the bony fragments at rest and in apposition. The patient should be kept in bed if there has been more than very slight injury to the soft tissues.

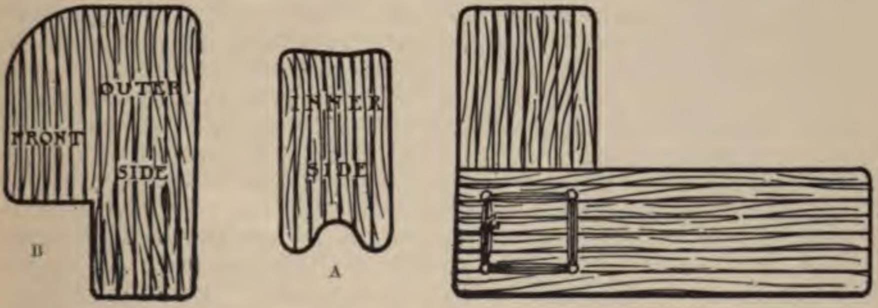
True *fracture of the acromion* is rare, although scapulæ with separated acromial processes are found in every pathological museum, for the majority of the specimens are examples of separation of the acromial epiphyses. In a case of true fracture of the acromion there is a slight flattening of the shoulder with a gap in the bone at the seat of injury, caused by the deltoid drawing the outer fragment downwards. Crepitus is not obtained until the arm is raised sufficiently to bring the two broken ends together. Local pain and tenderness are well marked, and there is impaired movement of the arm, especially in abduction. Repair takes place by fibrous union if there is much displacement, though it may be bony if the broken piece can be maintained in good apposition.

The *treatment* consists in placing a Stromeyer's cushion between the elbow and the side of the body, the arm being afterwards carried across the chest until the deltoid is completely relaxed. A plaster of Paris dressing passing round the chest and over the sound side shoulder fixes the injured limb. If it is undesirable to use plaster of Paris the arm should be supported by a large arm-sling to raise the elbow, and fixed by a bandage carried round the body, a gutta-percha or poroplastic cap being moulded to the shoulder. Passive movement may be commenced a fortnight after the accident, but the surgeon should not give too hopeful a prognosis, for many cases of apparent fracture of the acromion are associated with osteoarthritis.

THE HUMERUS.—A broken arm is a very common accident at all times and in every station of life. Some of the varieties are considered under various headings (see "Elbow Joint," "Shoulder Joint"). It remains, therefore, to consider the fractures of the shaft of the bone. These fractures are either primary or secondary. The primary fractures are caused by direct violence, as by blows; by indirect violence from falls upon the arm and hand, or they may be the result of muscular action. Secondary fractures are not uncommon in persons affected with scirrhus of the breast when they are due to secondary deposits of cancer, which so weaken the bone that it is broken by a very slight degree of force. The primary fractures are generally simple, and they are either oblique, transverse, or comminuted, the amount of displacement varying with each variety being least in the transverse and most in the oblique form. The displacement varies, too, with the position of the fracture, so that surgeons have been inclined to classify fractures of the shaft of the humerus into fractures above and fractures below the insertion of the deltoid, owing to the traction exercised by this muscle. Although the nature of the injury is usually quite obvious, for there is pain, increased mobility, deformity, and shortening, it is sometimes difficult to recognise a broken arm, as in the case of a rickety child, where the fracture is often transverse without rupture of the thickened periosteum and the bone is already deformed by rickets.

The *prognosis* is good in the vast majority of cases, though the patient should be warned that there is some danger of non-union and of nerve complications. It may be assumed as a broad rule for the prognosis of repair that the more marked the crepitus the more likely is there to be bony union, whilst great mobility at the seat of fracture, with only a little muffled crepitus, points to a complete separation of the two ends of the broken bone and to the intervention of a piece of the surrounding muscle or fibrous tissue. These conditions are likely to lead to the formation of a false joint unless the ends of the bones are exposed, the intervening tissue is removed, and the fragments are secured to each other by mechanical means. The musculo-spiral nerve is so closely attached to the humerus in the musculo-spiral groove, that it may be injured either at the time of the accident by the violence causing the fracture or by the spicules of broken bone, or, as is more often the case, by the nerve becoming involved in callus during the process of repair. As a rule the paralysis remains unnoticed until the splint is removed, when the patient complains of wrist-drop with impaired power of supination, accompanied by atrophy of the extensor muscles and loss of sensation in the skin supplied by the radial nerve. The application of electricity may be serviceable in some of the slighter cases, but unless improvement is marked and rapid it is advisable to cut down upon the humerus at the seat of fracture and free the nerve from its bony sheath.

The *indications for treatment* of a fracture near the middle of the shaft of the humerus are to fix the arm—whilst slight extension is kept up—in such a manner that the forearm may be flexed and slung without obstruction to the circulation at the elbow. This is best effected by applying a well-padded rectangular wooden splint along the inner side of the arm from the axilla to the finger-tips, with a Gooch's kettle-holder splint upon the outer side. Chiene recommends the following simple and satisfactory method:—"Take two Gooch splints, shaped as in Figs. 8 and 9, pad them well, and bind them on with a roller bandage. The notch at A (Fig. 9) is to avoid pressure upon the internal condyle, and the one at B (Fig. 8) fits the forearm, which is held at a right angle to the upper arm by means of a rectangular splint, also made of Gooch, as in Fig. 10, which reaches as far as the back of the wrist. The rectangular splint can be made easily by boring four holes and threading strong string, as in the Diagram." The bandage must be applied over the splint and to the forearm first with the elbow bent at a right angle. The actual seat of fracture should not be covered with the bandage. This bandage should end above the elbow. A second bandage should then be applied round the splint and humerus above the fracture, and whilst it is being applied the limb should be gently extended until it is of the same length as its fellow. The measurements are made from the lower and back part of the acromion, where a sharp edge of bone can be felt to the point of the olecranon. As soon as the splints have been applied, the hand and wrist are to be slung in a small arm-sling, that the weight of the forearm may help to maintain extension, and so diminish the tendency to shortening.



Figs. 8, 9, 10.—Simple splints for fractures of the shaft of the humerus made out of Gooch's "kettleholder" splinting. (Chiene and Dobie.)

There is so real a danger of delayed union going on to the formation of a false joint after fracture of the shaft of the humerus, that it is best to keep the arm in splints until complete repair has taken place, that is to say, for three weeks in children, and a month or five weeks in adults. In cases of delayed union the arm must be again secured for a further period of five weeks, complete immobility being maintained by means of a poroplastic or plaster of Paris dressing, fixing the shoulder as well as the elbow. Some further operation is necessary when it is clear that the humerus is not going to unite in spite of prolonged rest. Many methods have been employed with success, but perhaps the most usual one is to expose the ends of the bone, to refresh them by removing the fibrous tissue which covers them, and by then uniting them in the best possible position either with stout silver wire or by means of steel screws. Bony union takes place in rather more than half the cases treated by operation.

Supracondylar Fracture.—Fractures of the lower end of the humerus are very numerous, but the majority involve the elbow joint and are discussed already (vol. iii. p. 225, *q.v.*) A transverse fracture of the humerus just above the condyles is not a very rare accident after a fall upon the outstretched hand, or, less often, from a blow upon the bent elbow. The line of fracture is oblique from behind forwards and downwards when the arm has been broken from indirect violence with the elbow straight; whilst the line of fracture runs obliquely from in front downwards and backwards when the accident has happened with the elbow bent. In either case there is well-marked shortening of the arm, the shortening disappearing when the arm is pulled upon, though it reappears as soon as the extension is relaxed. It is often impossible to bend the arm beyond a certain point, because the ends of the two fragments interlock.

The injury is soon followed by swelling, which makes a differential diagnosis difficult; but if the patient is seen directly, it is easy to distinguish a supracondylar fracture of the humerus from a dislocation backwards of the radius and ulna, by the fact that the internal and external condyles of the humerus and the tip of the olecranon maintain their normal relation to each other, that is to say, the point of the olecranon lies vertically below a line joining the condyles when the elbow is bent to a right angle, so that a ruler placed on the back of the humerus does not touch the tip of the olecranon when the arm is bent to this angle. In children this accident has to be distinguished from a separated epiphysis. "The crepitus," says Mr. Poland, "in separation of the epiphysis is more 'muffled'; the lower end of the upper fragment has a greater breadth than in fracture: the line of separation is nearer the end of the bone, and the anterior projection of the shaft of the humerus is on a level with the fold of the elbow, whilst in fracture it is usually above it: the anterior projection, too, in a separated epiphysis has a rounded extremity unlike the projection of the sharp end of a fracture." Many accidents attend a supracondylar fracture of the humerus in addition to impaired movement at the elbow. The pressure of the one end of the broken fragments may obstruct the circulation through the brachial artery, and may even cause gangrene, an examination of the radial pulse must therefore be made from time to time. The median nerve may be injured by the same pressure, or the ulnar and musculo-spiral nerves may, at a later time, be involved in the callus produced during the process of repair, whilst there is often permanent deformity of the elbow, which is most obvious in full extension of the arm known as cubitus varus.

Every variety of position, from extreme flexion to complete extension of the arm, has been advocated in the treatment of supracondylar fracture of the humerus. Prof. Chiene, than whom we could have no higher authority, recommends that the arm be placed vertically upon the chest wall, whilst the forearm is fully flexed. A figure-of-eight bandage is then applied to the elbow and the arm is bandaged to the side. The French surgeons maintain the arm in full extension, saying that by this means the fragments are kept in the best apposition. For my own part I prefer an intermediate course, and after reducing the fracture, under an anæsthetic if necessary, I bend the elbow, put the arm midway between pronation and supination with the thumb uppermost, and place a well-padded rectangular splint of plaster of Paris along the inner side of the arm. The splint reaches from the axilla to the tips of the fingers and is kept in position by a roller bandage of linen, the arm being slung across the chest by a large arm-sling. Details of making and applying such a plaster of Paris case are given at vol. i. p. 459.

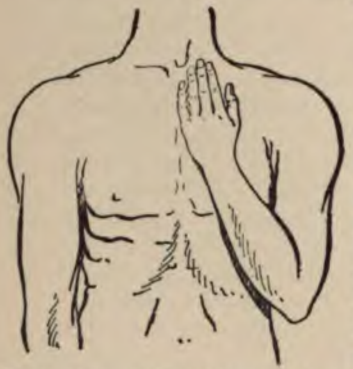


Fig. 11.—The position in which the arm is to be maintained in a case of fracture of the lower end of the humerus. (Chiene and Dobie.)

Passive movement should be commenced early, a point emphasised by

Mr. Joseph Bell, who teaches that fractures near the elbow in young people should be kept quiet for a period corresponding to the age of the child, a day for each year, *e.g.*, a child of five years for five days, of ten years for ten days. Slight movements of flexion and extension are carried out at the elbow joint, care being taken that no movement is allowed at the fracture, and that no pain is caused. Voluntary movements may be allowed at the end of a month, but if there is much swelling or stiffness the arm should be shampooed daily from the twentieth day.

Intra-articular fractures of the condyles have already been considered (vol. iii. p. 225), but a portion of the inner condyle may be broken off without injury to the elbow joint. Such an epitrochlear fracture, or fracture of the internal epicondyle, may be the result of a direct injury to the inner side of the humerus, or, less commonly, to muscular action or a fall upon the hand. It occurs chiefly in young adults, and is often of the nature of a separated epiphysis. I have recently had two cases under my care, both occurring as the results of accidents in the football field. The detached fragment is dragged downwards and either forwards or backwards by the flexor muscles which are attached to it, and by the pronator radii teres. There is much loss of function, passive movement is restricted, and the detached piece of bone can be moved independently. The accident may be associated with dislocation of the elbow: the ulnar nerve may be injured either by direct pressure of the condyle, or by the formation of callus; or "the carrying angle" of the arm at the elbow may be lost as the result of adduction of the forearm.

The treatment consists in semi-flexion of the elbow, wrist, and fingers to relax the flexor muscles; the epicondyle is then to be replaced as far as may be possible, being kept in position by a pad and strapping, and an anterior angular splint of plaster of Paris or metal is applied to the arm and forearm. The hand and wrist are alone slung.

Passive movement should be begun on the seventh to the tenth day, but it must be very gentle; the splints may be dispensed with upon the fourteenth day, when they are to be replaced by a sling. The detached piece of bone must be resected if the elbow be dislocated and the epicondyle prevents reduction; whilst the existence of nerve symptoms would lead the surgeon to expose the ulna at the seat of injury.

THE RADIUS AND ULNA.—Both bones of the forearm may be broken by direct violence when the fracture is at the same level in the two bones; by indirect violence, when the radius is usually broken at a higher level than the ulna; or by machinery accidents, when the bones are often broken at several different places after the arm has been drawn round a revolving wheel. Greenstick fractures of both bones are often seen in children who have fallen upon their hands.

Except in greenstick fractures there is usually no difficulty in recognising the nature of the injury, for the pain, deformity, loss of power and crepitus, are sufficiently characteristic. The prognosis is good, but the surgeon must bear in mind that in no other fracture is gangrene so common and so insidious, that impairment of supination and pronation frequently occurs, and that delayed union which may end in non-union may follow any imperfect fixation of the limb. The impairment of movement is due to several causes. It is sometimes caused by a rotatory displacement of the upper part of the radius, when the bone is broken above the insertion of the pronator radii teres, for the upper fragment is then supinated by the biceps, whilst the lower part of the bone is maintained in a semi-prone position. The power of full supination is lost if the bone heals in this position. In oblique fractures there is a good deal of overriding of the ends of the bone, and there is often some angular displacement. Crossed union of the broken ends of the bones may therefore easily occur, that is to say, the end of the radius may become adherent to the end of the ulna and *vice versa*, or the interosseous space at the seat of fracture may be filled with callus, and the usefulness of the limb is much lessened.

The *treatment* consists in carefully reducing the fractures by extension and counter-extension of the arm, care being taken that the interosseous space is maintained at its greatest width, *i.e.* in full supination when the fracture is in the upper third of the bones, and midway between supination and pronation when the bones are broken at any point in the lower two-thirds of the arm.

When the bones are broken in the upper third the arm is put up in full supination by means of a plaster case. The plaster case is made by taking a pattern in paper (see Fig. 12) from the sound side, in such a way that the splint will extend from the armpit, along the back of the arm, over the elbow bent at a right angle to the finger-tips. The pattern is laid upon a piece of ordinary house flannel of two thicknesses, over which a piece of lint is placed. The house flannel and lint are cut to the pattern, the inner lining of lint being sufficiently large to allow of its being wrapped over the edges of the flannel. The flannel is saturated with plaster of Paris paste, made by mixing one pound of freshly-burnt plaster of Paris with one pint of water, the excess of plaster being wrung out of the flannel. The lining of lint is then put in, and the case is bandaged to the arm, which must be held firmly until the plaster has set, that is to say, until the case gives a ringing metallic sound upon being struck. The arm is suspended by a sling reaching from the hand to the elbow. When the bones are broken in the lower two-thirds the same care must be taken to set them accurately, or if the fracture be greenstick, the bones must be straightened forcibly, but instead of the plaster case, two well-padded, straight wooden splints must be applied. The arm is held midway between pronation and supination, so that the thumb looks uppermost, for in this position the radius and ulna lie parallel to each other and at some distance apart, the splints are applied to the front and back of the forearm, and reach from the elbow to the back of the wrist, and from the bend of the elbow to the end of the metacarpal bones respectively. Each splint should be an inch wider than the transverse diameter of the arm to prevent the two bones being squeezed together by the bandage, but not wider, or movement will be allowed between the splints. The two splints are fixed in position by a strip of adhesive plaster applied spirally, a bandage is then put on, and the arm is supported by a large arm-sling, reaching from the elbow to the hand. Care must be taken that the inner splint does not press into the bend of the elbow, and the arm must be examined from time to time to see that gangrene is not taking place, for as the onset is sometimes painless, the statements of the patient are quite unreliable upon this point.

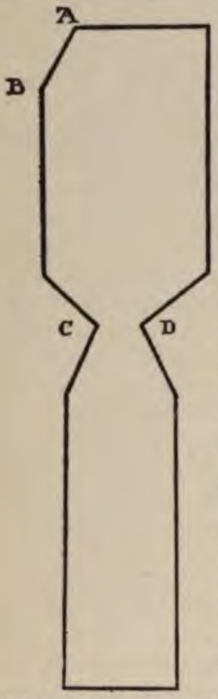


Fig. 12.—Diagram of a pattern for a plaster of Paris case for the back of the left arm. The piece AB is cut away for the axilla. The indentations at CD are to allow the elbow to be bent at a right angle.

The arm should be kept untouched in splints for a fortnight. It may then be taken down and passive movements of the wrist and elbow may be made daily, the splints being afterwards reapplied until the end of the third or fourth week, when the bones should be strong enough to allow the arm to be supported in a sling only. Unless the patient has to use his arm for especially laborious work he may be allowed to resume his occupation at the end of the sixth week from the injury.

The Ulna.—Fractures of the olecranon and of the coronoid process of the ulna are considered elsewhere (vol. iii. p. 228).

Fracture of the shaft of the ulna is generally caused by a blow upon the arm, more rarely it is the result of indirect violence, and occasionally it has been caused by muscular action. In children the fracture may be subperiosteal and incomplete. The special dangers attending the injury are the probability of the fracture becoming compound owing to the subcutaneous position of the shaft of the ulna and the tendency of the radius to become dislocated forwards, outwards, or least often backwards when the ulna is broken in the upper third. The dislocation is usually an immediate result of the injury, but it may be secondary to non-union of the broken ulna, or to the formation of exuberant callus during the process of repair. The lower fragment is drawn towards the ulna by the pronator quadratus, and is often displaced backwards, forwards, or laterally by the injury causing the fracture. The upper fragment, too, may be displaced either inwards or outwards, for there is a little lateral movement at the elbow, but the main displacement is forwards. The injury is not difficult to recognise when it occurs in the subcutaneous part of the bone, whilst the limited flexion, pronation, and supination with pain felt most severely at one spot, enables a diagnosis to be made when the fracture has occurred in the upper third of the bone.

Care must be taken in setting the fracture to reduce the lateral displacement of the lower fragment of the ulna towards the radius, and not to overlook any dislocation of the head of the radius. The lateral displacement is remedied by pressing the fingers and thumb between the two bones. The arm is then bent to a right angle, and is secured in a plaster case moulded to the arm and forearm in the manner already described. The arm is then slung in a large arm-sling for three weeks.

The prognosis is good, though the excessive formation of callus sometimes limits the movements of the elbow after a fracture of the ulna in the upper third. When the fracture is incomplete the radius is a sufficient splint, and the arm need only be slung. The early and gentle application of massage from the fourth day onwards is often serviceable in reducing the pain and swelling in these cases of greenstick fracture. When the injury is associated with a dislocation of the radius the elbow must be maintained at an acute, instead of at a right, angle, passive movements being commenced at the end of a fortnight.

Fractures of the Radius.—Fractures of the head and neck of the radius are considered on vol. iii. p. 229, whilst Colles' fracture is discussed under "Wrist Joint."

Fractures of the shaft of the radius alone are less common than fractures of the ulna, but like them are usually caused by direct violence. The displacement varies according to the position of the fracture: when it is above the insertion of the pronator radii teres, and below the insertion of the biceps, the upper fragment is rotated outwards, whilst the lower fragment is rolled inwards; but when the fracture is in the lower third the upper fragment is drawn forwards by the biceps, and is rotated internally by the pronator radii teres, whilst the lower fragment is pulled inwards by the pronator quadratus and the supinator longus.

The result, therefore, is often an angular deformity. The diagnosis of fracture of the shaft of the radius is easily established, if it is complete and unimpacted, by placing the fingers on the head of the radius, which can always be felt behind and directly below the external condyle of the humerus. If the radius be broken the head will not move when the hand and arm are gently pronated and supinated.

The treatment is the same as for fracture of both bones of the forearm (see p. 27), *i.e.* full supination when the fracture is above the insertion of the pronator radii teres, semi-pronation in all fractures of the middle and lower thirds. Repair takes place in a month, but the patient should be warned that pronation and supination may be impaired. Fractures of the various carpal bones are considered under the heading "Wrist Joint."

The Metacarpal Bones.—Fractures of the metacarpal bones are said to form only about one per cent of all the fractures occurring in the human body, the third and fourth metacarpals being broken more often than the first. The fractures are caused either by direct violence, as in fighting, the falling of a weight, or the kick of a horse, much more rarely by indirect violence, as by hyper-extension or over-flexion of the bones. Although complete fractures are the most frequent, it is by no means uncommon to see fissured fractures, and fractures where only a part of the bone has been torn off. The complete fractures are most common at the point where the bone is thinnest, that is to say, just above the middle. Prof. E. H. Bennett has recently drawn attention to a form of incomplete fracture which is not unusual in the metacarpal bone of the thumb, where after a fall upon the palm of the hand a fracture is caused which passes obliquely through the base of the bone (Fig. 13), detaching the greater part of the articular facet with that piece of bone supporting it which projects into the palm of the hand. The amount of displacement in this form of fracture is slight, but it consists in a subluxation of the thumb backwards. The displacement in the complete fractures is characteristic, unless it is masked by the swelling, for the proximal fragment is fixed whilst the distal end of the bone is tilted by the flexors of the fingers so that it projects upon the dorsum of the hand. Much more rarely the displacement of the distal fragment is into the palm of the hand, or even laterally. When a metacarpal bone is broken close to the head, the distal end is carried forwards into the palm of the hand, and the accident may be mistaken for a metacarpo-phalangeal dislocation.

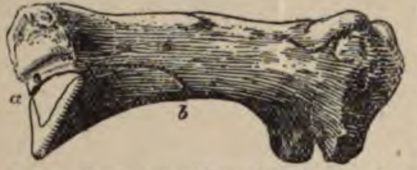


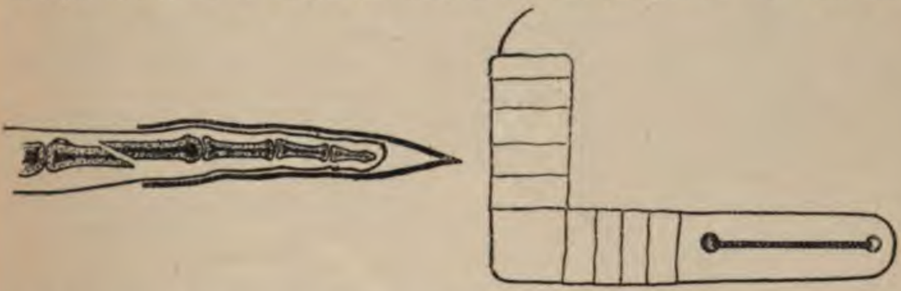
Fig. 13.—The metacarpal bone of the thumb to show the line of injury in Bennett's fracture of "the stave of the thumb."

The displacement and the increased mobility at the seat of fracture, combined with the sharp local pain, which is increased when pressure is made upon the head of the metacarpal, are sufficient to establish the diagnosis in cases of complete fracture. It is more difficult to recognise the injury when only a small piece of bone is broken off, but a radiograph will determine the nature of the injury.

The *treatment* consists in securing the hand for a fortnight to a well-padded Carr's splint, which can be made easily by screwing six inches of a broom handle obliquely on to the end of a straight wooden splint, so that the radial side is longer than the ulnar, the injured arm being afterwards suspended in a large arm-sling. A simpler plan is to bandage the closed fist over a pad of wool of sufficient size to completely fill the fist, but I do not think that this method is either so comfortable or gives such good results. In oblique fractures, where the surgeon considers it necessary to employ extension, a rectangular splint should be applied to the arm, so that it reaches well beyond the tips of the fingers; sticking plaster may then be applied to the injured finger, as in Fig. 14; the plaster is attached to a piece of elastic tubing, which is then carried through a hole in the splint and fixed to the back of the splint (Fig. 15).

There is often a little shortening after an oblique fracture of a metacarpal in spite of all treatment, but it is not of much importance, as it does not interfere with the movements of the hand. Repair is often very slow, though non-union is a rare result, and the formation of exuberant callus sometimes hinders the free movement of the fingers if it displaces the long flexor tendons. The prognosis of a simple fracture of the metacarpals is good, therefore, as regards union, but in many cases in Bennett's fracture of the metacarpal bone of the thumb there is often a lame and useless hand for many months after the injury.

THE PHALANGES.—The proximal and ungual phalanges of the fingers are more often broken than the intermediate ones, and as the injury is always the result of direct violence, it is often compound. The line of fracture in the distal phalanges may be vertical. In simple fractures, and in every case where the thumb is injured, attempts should be made to ensure repair, but in compound comminuted fractures of the ungual phalanges it is better to amputate at once, because union will probably result in a stiff finger-tip anchylosed at an awkward angle after very prolonged treatment. It is usually sufficient in simple fractures to adjust the fragments and keep the finger straight by applying a well-padded splint of wood, millboard, or gutta-percha, reaching along the palmar surface from the wrist to the finger-end. Passive movements may be commenced on the tenth day.



FIGS. 14, 15.—Method of applying a splint in a case of oblique fracture of a metacarpal bone. (Chiene and Dobie.)

THE PELVIS.—Much ingenuity has been displayed in constructing elaborate classifications of the various fractures to which the pelvis is liable. But it is sufficient for a practical work to divide them into simple fractures and complex, the simple fractures being those in which a portion of the bones are separated without other serious injury; the complex fractures being associated with dislocations of the bones upon each other, or of the femur upon the ilium, with injuries to the contained viscera and urethra, or with epiphyseal displacements. A fractured pelvis is usually caused by some crushing force, as in railway accidents, when the patient has been caught between a moving train and the platform, or between the buffers of two carriages; or by a fall of earth, stone, or coal, as in mining or quarry accidents. The pelvis has been fractured occasionally by indirect violence, as in those cases where a patient has fallen upon his feet, and the force has been sufficient to drive the head of the femur through the acetabulum, thus splintering the innominate bone.

The *signs* vary with the nature and severity of the injury, but there is always pain and loss of power in the lower extremity. When the innominate bone is broken there is increased mobility at the seat of injury, and crepitus can be obtained by gently rocking the pelvis. Injuries to the ischium can be readily explored through the rectum or vagina whilst the tuberosity is moved beneath the skin. Rotation of the femur will cause crepitus when the acetabulum is broken.

The special dangers attending fractures of the pelvis are rupture of the urethra, laceration of the bladder either within or outside the peritoneum, tearing of the rectum, and injuries to the spinal cord, the larger nerve trunks of the lumbo-sacral plexus, the iliac arteries, or the iliac veins. Laceration of the membranous portion of the urethra is the most usual complication of fractures and separations at the pubes. It is of such importance to treat this injury at once, and before there has been any extravasation of urine, that a catheter should be passed in every case of fracture at the front of the pelvis, and if it is impossible to reach the bladder an external urethrotomy should be performed.

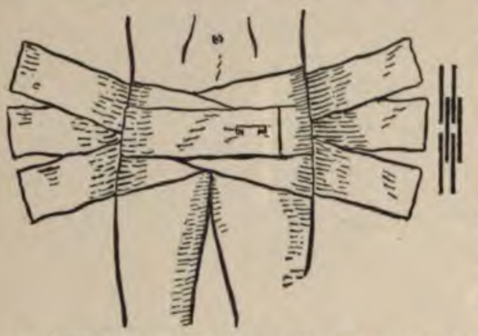


FIG. 16.—Method of applying a Scultetus' many-tailed bandage in a case of fractured pelvis. (Chiene and Dobie.)

The *treatment* necessarily varies with the nature and severity of the injury, but it always requires that the patient should be kept in bed for a lengthened period. A many-tailed bandage, made in two layers and folded as in Fig. 16, is often a sufficient support. A loop of bandage should be passed under each thigh and pinned to the lower limit of the many-tailed bandage to prevent it slipping upwards. In bad cases double extension with the legs apart must be applied, and in all cases a tripartite mattress (Fig. 17), the central portion of which may be removed for the use of the bed-pan, is a most useful addition to the comfort of the patient, and it is indispensable when a fracture of the sacrum has caused paralysis of the lower extremities, of the bladder, and of the rectum. A patient may be allowed to get about on crutches in the simple cases at the end of six or eight weeks, his pelvis being protected by a poroplastic splint.

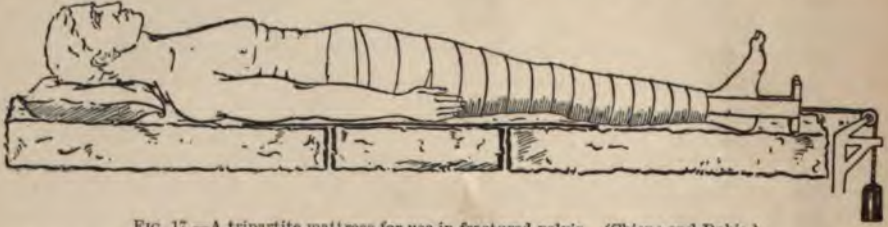


FIG. 17.—A tripartite mattress for use in fractured pelvis. (Chiene and Dobie.)

The prognosis varies greatly. The injury is often fatal, either at once from the shock and from the important structures involved, or after a long period of illness from the profuse suppuration of the connective tissues which are so abundant within the pelvis. Even in the least serious fractures, where bony union has taken place in six or eight weeks, there is often an impairment of movement, and the patient remains lame for life.

THE FEMUR.—Fractures of the surgical neck of the femur, including the intra-capsular and extra-capsular fractures, impacted and unimpacted, are considered under the article "Hip-Joint," whilst in the same manner fractures of the lower end of the femur, involving or near the knee, will be discussed under the heading "Knee-Joint."

Fractures of the shaft of the femur are divided conveniently into those which occur below the trochanters and those of the middle third. They occur both in children and in adults, but in adults they are seen more often in men than in women.

Fractures in the upper third of the shaft of the femur are nearly always the result of indirect violence, though they are due occasionally to muscular action. They are always very oblique and are often spiral, but in children a fracture through the shaft of the femur may be transverse or of the greenstick variety.

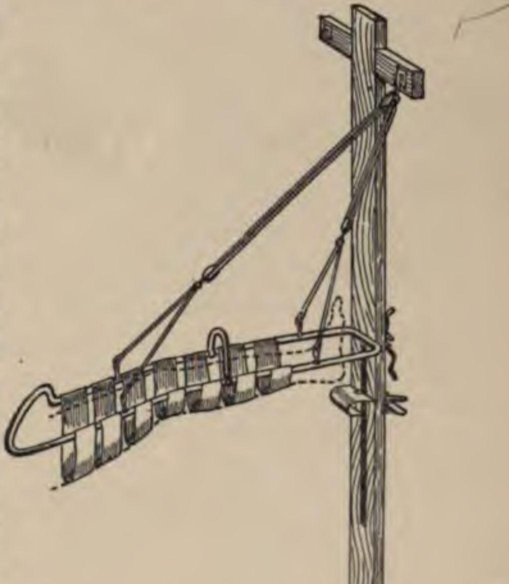


FIG. 18.—A fractured thigh treated by Hodgen's splint.

The *signs* of a subtrochanteric fracture are sufficiently characteristic to leave very little doubt as to its nature in the majority of cases. The pain deformity, unusual mobility at the seat of fracture, and the crepitus are well marked, whilst there is much shortening. The displacement is considerable, and is a troublesome factor in treatment. The short upper fragment is tilted forwards at a varying angle to the pelvis by the action of the psoas and iliacus muscles, and is at the same time everted and drawn outwards by the external rotator and gluteus minimus muscles. The lower fragment is drawn upwards by the rectus and hamstrings, whilst it is rotated outward by the weight of the foot and the adductors. The direction of the obliquity often causes the lower fragment to lie along the inner side of the upper fragment.

The *indications for treatment* are to approximate the two ends of the bone, and to maintain the lower part of the shaft in an abducted position. These indications are followed by flexing the thigh, by traction, and by allowing it to lie somewhat upon its outer side during the process of repair. A Hodgen's splint (Fig. 18) is therefore the most satisfactory appliance for subtrochanteric fractures. It consists of two rods of iron slightly bent at the level of the knee, the two bars being connected at the lower end by a straight bar and at the upper end by a curved one. The splint reaches from the anterior superior spine of the ilium to three inches below the external malleolus on the outer side of the leg, and from the adductor longus tendon to an equal distance below the internal malleolus on the inner side of the limb. It is applied to the front of the leg and thigh, so that the limb is slung between the lateral rods by a series of strips of house-flannel which overlap each other, the ends of each strip being separated, stitched, or pinned round the sides of the splint. Extension is made by a weight, pulley, and strapping, in the manner to be described immediately, and the whole apparatus is slung at a convenient angle, as is seen in Fig. 18. Under many conditions it is inconvenient or impossible to use a Hodgen's splint, and recourse must then be had to the double inclined plane (Fig. 19). The angle should be as obtuse as possible, and only so much bandage should be applied as is sufficient to keep the limb at rest in a good position. The splint should be kept on for six weeks or two months, the thigh being massaged after the twentieth day. When the splint is removed the patient must be careful of his leg for some time, as it is not unusual for the thigh to be refractured as the result of a false step or a slip. A plaster of Paris case may be applied to the thigh for a month after the splint has been removed, the patient learning to walk in the meantime upon crutches. In every case he must be warned that the shortening may be so considerable as to cause him to limp, but there is usually good bony union. In cases of angular union, and when the limb is greatly crippled, the question of resection of the end of the bone may be reasonably entertained. The surgeon must, of course, deal with each of these cases on its merits, but unless he be skilful, experienced, and in a position to carry out the operation without any chance of suppuration, he had better not undertake it even in the most favourable case.

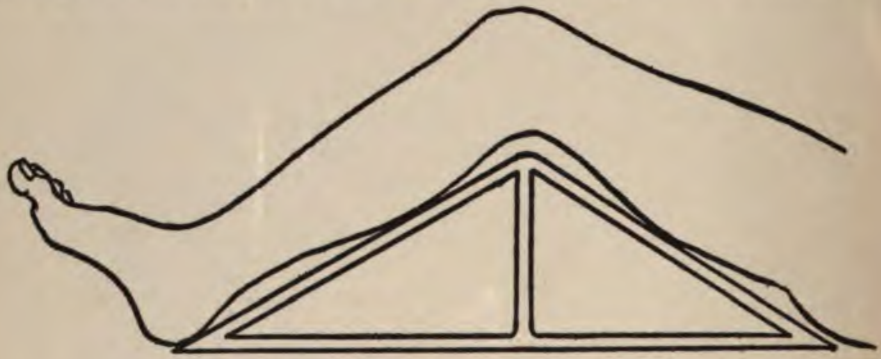


FIG. 19.—A double-inclined plane for use in certain fractures of the femur.

Fracture of the Middle of the Shaft of the Femur.—Fracture of the middle of the shaft of the femur is a common accident both in adults and in children, either as a result of indirect violence or less frequently from blows upon the thigh or the passage of a wheel over it. The line of fracture is usually oblique from before backwards, so that the lower fragment passes behind the upper, which may be so sharp as to perforate the muscle and skin, thus rendering the injury compound. The obliquity may be so great as to make the line of fracture almost vertical, or it may become spiral by following the arrangement of the fibres of the osseous tissue at this part of the femur. The union of various lines of fracture may detach large splinters of bone, though the fracture is not truly comminuted. On the other hand, greenstick, subperiosteal, and transverse fractures are by no means rare in children, and true comminuted fractures may occur in them, as a result of scurvy, without any appreciable violence.

The *signs* of a fractured thigh are generally sufficiently distinct. There are pain, loss of power over the limb, abnormal movement at the painful spot, and crepitus, whilst the injured leg is materially shorter than the sound one and is very completely everted. In making comparative measurements of the length of the two limbs care must be taken that the patient is lying with his two iliac bones upon the same level, for inaccurate results will be obtained if the pelvis be tilted. The measurements are made from the anterior superior spine of the ilium to the tip of the external malleolus. The diagnosis is often extremely difficult in children with incomplete fracture, and the nature of the injury is often entirely overlooked. The prognosis is good in simple fractures of the middle of the shaft of the femur, but the surgeon should remember that delayed and non-union are not uncommon, that it is the most difficult thing in the world to get union without shortening when the fracture is very oblique or comminuted, and that a passive synovitis of the knee takes place in nearly every fractured thigh, even though the bone is broken in the upper or middle third.

Treatment.—There are very many different methods of treating a simple fracture of the thigh, but here, as always, the simplest is the best, and the application of a long external splint with a weight extension will usually answer every purpose. In the first place, care must be taken in moving a person who has fractured his femur to prevent the simple fracture becoming compound, and this accident is the more likely to happen the nearer the injury is to the knee. The limb should therefore be immobilised by securing a long external splint and a shorter internal one to the injured limb. A lath or broom handle reaching from the armpit to the sole, with an umbrella or walking-stick along the inner side of the thigh, will answer this purpose temporarily, if they be secured with handkerchiefs tied round the groin, above the knee, below the knee, and at the ankle, the patient's legs being afterwards bound together for additional security. The patient will have to be kept in bed and nursed for several weeks, and he should therefore be placed at once upon a low and narrow iron bedstead provided with a good hair mattress, which is not likely to become uneven, and sand-bags should be placed upon either side of the leg until the surgeon is ready to reduce the fracture.

The leg and thigh should now be carefully washed, dried, and dusted over with oxide of zinc. A piece of stout moleskin strapping, 2½ inches wide and about five feet in length, is doubled upon itself, and a square piece of wood, whose diameter is equal to the width of the ankle at the malleoli, is secured in the centre of the strapping by four drawing-pins. The strapping is warmed and applied to either side of the leg, care being taken that it reaches two or three inches above the knee, and that it does not stick to the malleoli, for which purpose the lower six inches of strapping upon either side of the leg are rendered non-adhesive by guarding each strip with a second piece of strapping of the same width, so that the two adhesive surfaces are together. The two pieces of strapping running up the leg are kept in place by winding a second piece of plaster spirally round the leg. A cord is then passed through the centre of the wooden stirrup, which should lie quite square about three inches from the sole of the foot. A long external splint is now chosen, of such a length that one end lies comfortably in the axilla without exerting any pressure upon the armpit when the arm is by the side, whilst the external malleolus is opposite the centre of the hole in the splint which is made to receive it. The splint should have no foot-piece, but a flat cross-piece is screwed horizontally to the lower end to keep the splint steady. The whole splint must be well padded with tow, and it is then laid along the injured side of the patient, who is placed flat on the bed without any pillow under his head. Two assistants make steady extension upon the limb until the deformity at the seat of fracture disappears, the one taking hold of the thigh just above the knee and rotating it inwards, whilst the second assistant grasps the foot and ankle, which must also be rotated inwards to the same extent as the thigh. The surgeon secures the splint by applying a bandage round the foot and ankle in such a manner as to keep the foot at right angles to the leg, the bandage being afterwards carried up the leg as high as the tubercle of the tibia. A second bandage is then applied to the thigh above the seat of fracture, and a broad flannel bandage is carried round the chest and over the splint. The knee and the thigh in the immediate neighbourhood of the fracture are not bandaged, and it is best to apply the bandage to the foot and leg from without inwards to counteract the tendency to eversion. When a single outside splint fails to support the fracture it may be reinforced by a straight and well-padded wooden splint put at the back of the limb and reaching from the gluteal fold to the top of the calf. In other cases a piece of Gooch's "kettle-holder" splint may be cut to encircle the front and inner side of the thigh from the groin to the top of the patella. A small pulley is then fixed to the end of the bed, either by screwing it into a piece of wood fastened to the bed-foot, or by means of a standard with a movable arm standing just beyond it. The cord attached to the stirrup runs over the pulley and, for an adult, carries a weight of ten pounds. The foot of the bed is then raised about four inches by means of two blocks of wood, that the patient's body may act as a counterpoise to the traction of the weight. The injured limb is wrapped in a small blanket, a cradle is put over it, and the rest of the bed is made as usual. Attention must be paid during the after-treatment to the comfort of the patient, especially to any indication of chafing at the heel, the malleoli, or over the sacrum, and the surgeon should bear in mind that the effects of chronic alcoholism show themselves more readily after a fracture of the thigh than after any other fracture. The first effect of the weight is to overcome the muscular spasm, but as this lasts from twenty-four to forty-eight hours, sufficient time should be allowed to elapse before the lighter weight is replaced by a heavier one. In muscular subjects a much heavier weight will be required to secure a proper extension, whilst in children the extending force is generally calculated as two pounds for a child of two years old, increasing a pound for each additional year. The weight and pulley need constant attention, as the cord often jams and renders the extension useless.

In children, where the fracture is much less oblique than in adults, a plaster of Paris case applied to the front and back of the limb and an extension apparatus answer admirably, the Liston's splint being applied to the uninjured side for the purpose of making the child lie flat in the bed. Children who have incontinence of urine, very young children, and those who have both thighs broken, can often be treated satisfactorily without any splint, by keeping them flat upon their backs, and rigging up an extension apparatus consisting of a weight and pulley so situated above the bed that the thighs are flexed at a right angle with the body. Restless children who cannot be induced to lie straight and flat by any other means are best secured in a Bryant's or double Thomas's splint.

The duration of treatment varies with the age, weight, and general condition of the patient. Three weeks in bed is sufficient for a baby who cannot walk; for a child, a month with a further fortnight in a plaster of Paris case; but as age increases and the patient is heavier, a longer time is required, so that an adult will be four or five months before he can walk with ease. The wasting and the stiffness of the knee are the most troublesome factors in the convalescence, and these are combated satisfactorily by the employment of massage from the twentieth day onwards, or at a later period by the use of a hot-air apparatus. The splint may be permanently removed at the end of five or six weeks, but the patient should then be kept in bed until at the beginning of the seventh week he is allowed to get up with a plaster case upon his thigh. He must then learn to walk upon crutches, for too early walking upon a fractured femur causes bending of the bone, or it may again be broken.

The symptoms and treatment of inter-condyloid fractures of the femur and of fracture of the inner and outer condyles are considered under the heading "Knee, Diseases and Injuries of."

FRACTURES OF THE LEG.—Fractures of the upper end of the tibia necessarily involve the knee-joint, and are described under the article "Knee-Joint."

FRACTURES OF BOTH BONES OF THE LEG.—Fractures of the shaft of the tibia and fibula are the result of direct violence, when the fractures are either transverse or comminuted, and the two bones are broken at the same level; or they are caused by indirect violence, when the tibia is broken first, the fibula yielding afterwards and at a higher point. A forcible muscular contraction is occasionally sufficient to fracture the tibia, the fibula breaking subsequently when an attempt is made by the patient to bear his weight upon the injured limb. The tibia is especially liable to be broken by torsion, and in these cases the line of fracture is often spiral, or the lower fragment is comminuted whilst the upper fragment terminates in a sharp V-shaped end which often penetrates the skin, making the fracture compound.

The signs are usually so well marked that there is no difficulty in recognising the injury, though it is sometimes difficult to discover whether the fibula is broken as well as the tibia. The prognosis varies with the severity of the injury, except in children. Good bony union nearly always takes place in a simple transverse fracture where there is little or no displacement. But when the fracture is comminuted or oblique the repair is much less satisfactory, for it takes considerably longer, there is often impaired movement at the knee or ankle, and the patient may complain of rheumatic pains for many years afterwards. The course taken by a compound fracture which has become septic is still more tedious, and in unhealthy people is often dangerous. In children of two or three years old the simplest fracture requires as much care in its treatment as the most severe, for I have several times seen so intractable a form of non-union as a result of a simple fracture that amputation has been required.

Care during the first few minutes after a patient has broken his leg will often save months of trouble, for thoughtless bystanders often try to set a man upon his legs when he has fallen, or lift him so roughly or carelessly as to force the upper end of the broken tibia through the skin, thus converting a simple into a compound fracture. A doubtful injury to the leg should always be treated as a fracture, and the patient should be moved out of harm's way as gently as possible, some one supporting the injured limb and doing nothing else. An improvised splint should always be applied to the injured limb to steady it whilst the patient is being removed to his home or a hospital. The two parts of the broken leg are to be kept in the same straight line, and the injured limb is on no account to be allowed to hang down, nor should any attempt be made to drag off the boot or stocking. It is best to lay the leg on a pillow or on a folded coat, a handkerchief being tied round the leg and its support above and below the seat of fracture, or two lateral splints may be improvised out of a couple of walking-sticks, umbrellas, or even newspapers folded lengthwise.

The subsequent treatment varies with the severity of the injury and, perhaps more than any other fracture, with the school in which the surgeon was educated. Many surgeons content themselves in the simplest form of fracture with applying a plaster of Paris case at once; others keep the patient in bed a week to allow the swelling to subside and then employ a plaster case; whilst yet others, and I confess that my sympathies are with them, keep the patient in bed for a longer time and sling the leg in a back splint.

The plaster case consists of layers of house flannel shaped to fit the foot and leg (Fig. 20), and soaked in plaster of Paris in the manner described at p. 459, vol. i. An antero-posterior splint gives better support than the lateral ones usually recommended. The anterior splint is a narrow one reaching from the top of the patella to the end of the metatarsal bones; the posterior one is broad enough to support the leg both on its inner and outer sides, and it extends from the top of the ham to the web of the toes. Care must be taken whilst the splint is hardening to maintain the full length of the leg, to keep the foot exactly at right angles with the leg, especially to avoid eversion of the foot, and to see that the lower fragment is neither twisted nor tilted upon the upper fragment. The two parts of the plaster of Paris case are bound on to the leg with a figure-of-eight bandage applied before the plaster has set. If the plaster case is to be used at once it must be put on before the swelling has commenced, and the surgeon in charge of the case should himself hold the leg in proper position until the plaster has become thoroughly hard. He should remember that bullæ often form under the skin when the leg is broken, and that a case which fits accurately at first becomes sufficiently loose in the course of a week to allow of the fragments becoming displaced. He must therefore keep the patient under daily observation, and be prepared to apply a fresh case as soon as may be necessary. Massage may be commenced from the seventh day in simple cases, and should be particularly directed to the knee and ankle. The patient should be kept at rest for three weeks when both bones are broken, and he may then be allowed to walk upon crutches, the injured leg being kept in the plaster case for at least six weeks.

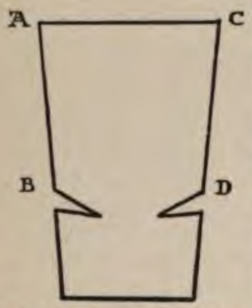


FIG. 20.—Diagram of a pattern for a plaster of Paris splint for the leg. The side AB corresponds to the length of the limb, AC to its circumference at the thigh and ankle respectively, the piece below BD being for the foot, and the indentations being for the ankle.

A certain amount of risk attends the immediate treatment of even a simple fracture by this method, and many surgeons therefore prefer to keep the patient in bed until the swelling has subsided before applying a plaster splint. The foot and leg must be kept steady and in a proper position for a week or more, and this is best effected by a back splint and suspension of the leg. For this purpose a well-padded metal back splint with a rectangular foot-piece is selected (Fig. 21). It should be sufficiently long to reach as high as the middle of the thigh, and broad enough to prevent any compression of the leg by the side splints and bandage. The splint must be bent to an obtuse angle opposite the knee to keep the joint in slight flexion, and thus to relax the muscles of the calf. The splint should be provided with two cross pieces with perforations of sufficient width to allow an ordinary leather rug-strap to pass through the hole on each side, one cross piece being situated just above the foot-piece, the other immediately below the knee. Two well-padded, straight wooden splints are also obtained, long enough to reach from the foot-piece to the knee, and about four inches in width. Provided with this apparatus, some small pads, a roll of strapping, bandages, safety-pins, a pair of rug straps, a "cradle," webbing straps with buckles, and a teacupful of starch paste, the surgeon proceeds to set the broken leg. The whole limb is shaved if necessary, and is thoroughly but gently washed with warm soap and water; it is dried and is dusted over with oxide of zinc. It is then raised from the bed by the surgeon, who takes a firm hold of the foot and of the calf, whilst an assistant slips the splint beneath it. The leg is then lowered on to the splint, the assistant putting a small pad beneath the tendo Achillis to prevent any pressure upon the heel. The foot is secured to the foot-piece by a roller bandage, one end of which is pinned into the padding on the inner side of the splint. The bandage is then carried between the padded foot-piece and the sole of the foot to the outer side, thence across the foot, round the back of the foot-piece, and again round the foot with a reverse. The surgeon now takes the foot-piece to which the foot is attached in his hands, whilst the assistant steadies the leg. Gentle traction is then made upon the foot until the displacement is reduced at the seat of fracture, and until the two fragments of the tibia are brought into good apposition. The foot and the lower fragment are also moved until the inner edge of the patella, the inner ankle, and the inner side of the great toe are in the same vertical plane when the patella looks directly upwards. A strip of adhesive plaster two inches wide is then carried spirally round the leg and splint just above the malleoli, a similar piece just below the knee, and a third piece round the thigh. A few turns of bandage with reverses are then put on at each of these places, leaving the fracture and the knee unbandaged, and a little starch paste is brushed over the bandages to keep them in position. The two lateral splints are applied to the leg and are kept in place by webbing straps, the cradle is put over the limb, and the splint is slung to it by the straps, so that it swings just clear of the bed.

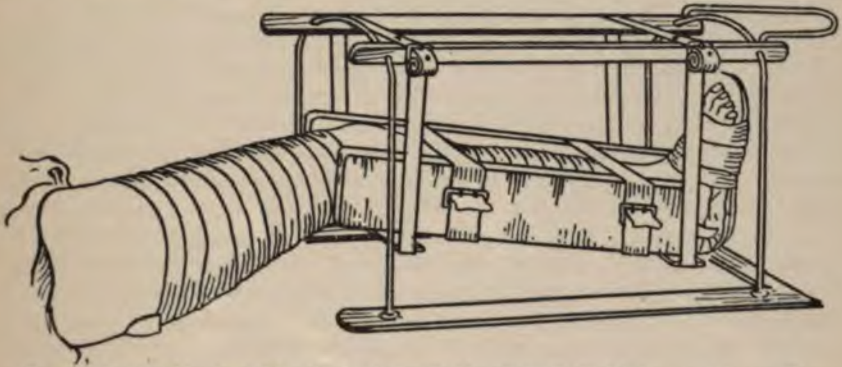


FIG. 21.—Fractured leg treated by a back splint with side splints, the limb being swung in a cradle.

Professor Chiene gives the following directions for making an improvised box splint when the more elaborate form here described is not available:—"Take two flat pieces of wood about half an inch thick, and between four and five inches broad, and long enough to reach from a little above the knee-joint to beyond the heel. A large sheet folded so that its breadth may be equal to the length of the splint is then prepared, and the splints are rolled up in it towards one another, beginning with one at either end of the sheet. Go on folding until the distance between the two splints, when vertical, may, along with a little padding, be just sufficient to enclose the limb. Pad well, and fix in position with three slip knots. Keep the foot at right angles to the long axis of the limb by means of elastic or a bandage, and place the leg upon a pillow. In oblique fractures of the tibia extension may be required to prevent shortening. In compound fractures it may be necessary to mould a splint to the leg, and rabbit wire netting folded into layers, to give it sufficient stability, with an old blanket as padding, can be fashioned into an excellent splint accurately fitting the injured limb."

When a box splint is used the surgeon must carefully watch the limb to see that the heel as well as the toes touch the rectangular foot-piece, as even a little equinus is very troublesome to the patient when he begins to walk again. He must also make sure that the heel pad is effective in keeping pressure off the heel, which easily gets sore. A well-applied splint, too, is comfortable, and will prevent the spasmodic starting which is often a very painful accompaniment of fracture. The surgeon should have no hesitation in taking down the whole splint and readjusting the fracture, if there is the least real ground for complaint, within the first few days of the injury, and he must always be ready to readjust the bandages to accommodate any unusual amount of swelling. The splint may be abandoned on the twenty-first day in favour of the plaster of Paris case before described, but massage of the leg and ankle may be commenced a week earlier. The process of shampooing is particularly valuable in these cases because the skin is apt to get dry and scurfy. It is better not to interfere with the bullæ, which are often large and numerous.

There are many cases in which it is impossible to get the fragments of bone into good apposition, and it is then justifiable to undertake a formal operation, exposing the ends, wiring them, and uniting the wound so that it heals by first intention.

Treatment of Compound Fractures of the Leg.—Compound fractures of the more simple variety should always be treated in the hope that they may heal aseptically, or at any rate with a minimum of constitutional disturbance. The surgeon should first wash and disinfect his hands, and when a sharp fragment of bone protrudes through the skin it must be thoroughly cleansed with hot soap and water. It is then bathed in a solution of biniodide of mercury, which is first of a strength of 1 in 500, and afterwards of 1 in 1000, the biniodide being somewhat more satisfactory than the perchloride of mercury. The fragment of bone is afterwards replaced—and the skin covering the shin is so thin and elastic that a comparatively large piece of bone leaves but a small hole when it has been returned to its normal position. The skin, both in the neighbourhood of the wound and for some distance round it, must be cleaned and thoroughly disinfected by scrubbing it with a nail-brush, after lathering with soap and water. It is then washed with turpentine or methylated spirit, soaked for two minutes in a solution of biniodide of mercury in methylated spirits (1 in 500), and afterwards dressed with a piece of cyanide gauze wetted with a solution of 1 in 2000 biniodide of mercury. A layer of cotton wool is placed over the gauze, and the whole is kept in place by a bandage. The limb is then treated in the same way as a simple fracture, and, in a healthy person, it often heals without further trouble.

It is better to enlarge the wound if there is any difficulty in reducing the fragment rather than bruise the skin and soft tissues by using violence. The fragment may be sawn off in extreme cases, and if there is much bleeding and laceration the patient should be placed under an anæsthetic and the parts thoroughly disinfected, the detached fragments of bone being removed, and the limb dressed in the manner just described. Severe compound comminuted fractures with extensive destruction of the soft parts and injury to the vessels and nerves require amputation.

Fractured Fibula.—The upper end of the fibula may be fractured by direct violence, as by the passage of a wheel over the leg, the tibia escaping injury; by indirect violence when the leg is so powerfully adducted as to put an extra strain upon the external lateral ligament, or by a sudden contraction of the biceps femoris muscle.

The signs of fracture are the altered position of the head of the fibula owing to the upward displacement caused by the biceps, and the impairment of function due to pressure upon, or injury to, the peroneal nerve at the point where it winds round the head of the bone.

The prognosis depends in great part upon the nerve lesions, which are either immediate or remote. Severe pain in the course of the nerve, with a burning pain referred to the foot, after an injury to the top of the fibula, is evidence that the nerve has been injured, whilst herpetic eruptions, ulceration, glossy skin, and the appearance of talipes equino-varus at a much later time, due to paralysis of the peronei and extensor muscles, would indicate that the nerve was seriously implicated, and would justify an incision in its course to ascertain the cause. Inasmuch as the synovial membrane of the tibio-fibular articulation sometimes communicates with the knee-joint, fracture of the upper end of the fibula is occasionally associated with some swelling and inflammation of the larger synovial membrane.

The treatment consists in relaxing the biceps to keep the two fragments in good apposition, and the leg should therefore be kept with the knee bent to a right angle for a fortnight or three weeks, though if an incision has to be made to explore the nerve there is no reason why the two fragments should not at the same time be united with silver wire. At the end of three weeks the patient may be allowed to walk with his leg and knee enclosed in a plaster case.

Fractures of the middle of the shaft of the fibula are generally caused by direct violence. They are either oblique, transverse, comminuted, multiple, or much more rarely incomplete. Simple fractures of the middle of the shaft of the fibula are sometimes very difficult to recognise, for the bone is well surrounded by muscle and the displacement is not great. Pain can be elicited locally by pressure, as well as by "springing" the fibula, that is to say, by holding the lower part of the leg steady whilst pressure is made upon the upper part of the outer side of the fibula. A skiagraph, however, will always make clear the nature of the injury. Here, if anywhere, the ambulatory treatment with massage may be employed. It consists in the application of a plaster of Paris case in two lateral portions. The foot must be kept at right angles, and care must be taken to see that the case continues to fit so long as it is used. Repair takes place in three weeks, but the patient may complain of neuralgic pain in the seat of injury. Fractures of the fibula within four inches of the external malleolus are considered under the article "Ankle-Joint," vol. i. p. 260.

Fractured Tibia.—Direct violence may fracture the tibia either at its upper end, when the line of fracture is transverse or more rarely vertical, or at any part of the shaft, when the fracture is usually oblique. The bone is sufficiently subcutaneous to enable a fracture to be detected easily if the patient be seen before the swelling has occurred, but at a later time the diagnosis is more difficult since the fibula acts as a splint and there is very little displacement. The prognosis is good. The treatment consists in keeping the patient in bed with his leg on a rectangular splint until the swelling has subsided. He may then be allowed to go about on crutches after a plaster of Paris case has been applied.

FRACTURES OF THE TARSUS (fractures of the astragalus are considered under the heading "Ankle, Diseases and Injuries of").—The os calcis is subject to several fractures. The bone may be crushed as a result of falls upon the feet, or severe injuries may be inflicted upon it by the forcible contraction of the powerful muscles attached to it; less often splinters of bone may be detached, or some of its bony prominences may be separated.

The foot is flexed at the ankle when the body of the bone is broken and the power of extension is lost; there is great pain in the tarsus, and crepitus can usually be felt. The width of the heel is increased, and the malleoli are nearer to the sole than on the sound side. The patient should be kneeling whilst the surgeon makes his preliminary examination. The diagnosis is often difficult, and the injury has been mistaken for a Pott's fracture and for a severe sprain. The prognosis is not very favourable, as some amount of flat-foot is nearly always left, and in severe fractures the ankle-joint may be involved. Treatment consists in keeping the foot and leg at rest in a plaster of Paris case for fully three weeks, massage being commenced at the end of a fortnight. When a violent contraction of the tendo Achillis has torn away a fragment from the posterior aspect of the os calcis it may be necessary to pin the fragment to the body of the bone, if it cannot be kept in place by flexing the knee and extending the foot.

The sustentaculum tali is occasionally torn off by forcible inversion of the sole of the foot. The accident is attended by pain, inability to walk, a sudden change from inversion to eversion of the foot, and shortening of the heel due to a slight displacement forward of the os calcis. Crepitus may be felt on the inner border of the foot. The prognosis is complicated by the subsequent tendency to flat-foot. The treatment consists in the application of a plaster of Paris case, with sufficient inversion of the foot to bring the two fragments together.

FRACTURES OF THE METATARSAL BONES.—The metatarsal bones are nearly always broken by direct violence, and it is said that the first and fifth metatarsals are more often fractured than the second, third, or fourth. The injury is either simple or compound, and the broken ends of the bone project beneath the skin on the dorsum of the foot. There is often so much swelling and bruising of the foot as to render the diagnosis difficult, though the localised pain renders the nature of the injury obvious.

In simple fractures the prognosis is good if care be taken to get union with the least possible displacement, but in compound fractures the severity of the injury may render amputation indispensable. The dirty habits of people liable to injury of the metatarsal bones make it necessary to be especially careful in disinfecting the feet when the skin is abraded, for the bruised tissues easily suppurate. A Cline's splint with the foot-piece at right angles applied to the outer side of the foot may be employed in these cases until the swelling has subsided.

FRACTURES OF THE METATARSAL PHALANGES.—The phalanges of the toes are broken by direct violence, and the fracture, except in the great toe, is nearly always compound. The pain and crepitus render the diagnosis easy. The prognosis is good in simple fractures, but extensive suppuration and necrosis may follow a compound fracture unless it be thoroughly disinfected immediately after the injury.

A gutta-percha splint may be moulded to the inner side of the foot in fractures of the great toe, whilst in fractures of the phalanges of the other toes the dressings applied to the wound are generally sufficient to keep the fragments in good apposition until union takes place. Every effort must be made to preserve the great toe after fracture, but in the other toes, when there has been comminution with much bruising of the tissues, it is sometimes more profitable to amputate at once.

LITERATURE.—Further details about Fractures and their treatment may be read in *A Treatise on Fractures and Dislocations*, by Lewis A. Stimpson, M.D., Professor of Surgery in Cornell University Medical College, New York. Henry Kimpton, 1899.—*A Practical Treatise on Fractures and Dislocations*, by Prof. F. H. Hamilton. Smith, Elder, and Co. Lond. 1884.—Helferich. *On Fractures and Dislocations*, translated by Jno. Hutchinson, Junior. The New Sydenham Society, 1899.—Prof. Ricard and Dr. Demoulin's article on "Lésions traumatiques des os," in Duplay and Reclus's *Traité de chirurgie*, 2nd edition, vol. ii. Paris, 1897.—Gurlt. *Handbuch der Lehre von den Knochenbruchen*, Berlin, 1885.—Lucas-Championnière. *Du massage dans le traitement des fractures*, Paris, 1895.—"The Treatment of Fractures by Simple Methods," by John Chiene, illustrated by E. Cyril Dobie, M.D. *The Practitioner*, vol. li. 1893, pp. 81-91.—C. H. Golding-Bird, "Diagnosis and Treatment of Spinal Fracture," *Brit. Med. Journal*, vol. i. 1900, p. 945.—Prof. Chiene and the Editor of the *Practitioner* have kindly allowed the following illustrations from this article to be reproduced in the present work:—Fig. 1, Fractured clavicle; Fig. 2, Fractured humerus; Fig. 3, Fractured metacarpals; Fig. 4, Fractured pelvis; Fig. 5, Fractured femur; Fig. 6, Fractured leg.

Fragilitas Ossium.

Fragilitas ossium or *Osteopsathyrosis* may be defined as an unnatural brittleness of the bones. The terms are employed to denote two different conditions—(i.) An abnormal brittleness of the bones occurring as a symptom in consequence, for example, of tumours in the bones, disuse, senility, joint disease, cachexia of malignant disease, syphilis, rickets, insanity, locomotor ataxia, scorbutus—*e.g.* several fractures recorded by Guthrie in a child æt. 2½ years, who had scurvy; and lastly, phosphorus poisoning, as in two workers in a lucifer-match factory, each of whom had "at different times both thigh bones broken in a ridiculously simple fashion" (Dearden). In such cases there is rarefaction of the bones, which are light and porous, and which may show deformity from repeated fractures. (ii.) Idiopathic Fragilitas ossium, a separate and distinct condition, not secondary to any pre-existing disease. The brittleness here is not accompanied, so far as is known, by any constant local alterations, nor does the chemical constitution of the bones satisfactorily account for their fragility. The bones, though very brittle, do not tend to bend, but in consequence of numerous fractures they become much deformed. The bones, and especially those of the lower limbs and the ribs, are liable to fracture after very slight violence, *e.g.* while throwing a cricket-ball (Willet), from a slight fall or blow, or even from mere movement, say, of the limbs, as in Spurrell's case. The disease is often, though not always, hereditary, and several members of the family may be affected, as in Hunter's cases. The first fracture often occurs during childhood, and subsequently fractures take place repeatedly. Stanley quotes the case of a girl aged fourteen, who had had 31 fractures. Dent records 27 fractures in a man aged twenty-nine, and in Tyrrell's case there had been 22 fractures—scarcely any long bone had escaped.

Diagnosis of the idiopathic form can only be made by excluding those conditions causing rarefaction and atrophy of bone. A history of the disease in the relatives is important, and it must not be confounded with osteomalacia. As Fragilitas ossium may affect the fœtus *in utero*, and the fœtus be born prematurely with multiple fractures, the disease has a certain importance from the medico-legal standpoint.

Treatment.—Union is usually rapid and firm. We have no means of checking or curing the disease itself.

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Frambœsia. *See* YAWS.

Friedreich's Ataxia. *See* PARALYSIS.

Frostbite. *See* GANGRENE.

Frontal Sinus. *See* NOSE.

Fungus Disease of India. *See* MYCETOMA.

Furuncle. *See* BOILS.

Furunculus Orientalis.—SYN.: Oriental Boil or Sore.

UNDER this heading must be described an affection which, under various names, is exceedingly common in some parts of tropical countries. Sooner or later almost every one residing in the tropics suffers from boils, either singly or in crops, but in some places boils appear to be endemic as well as epidemic, and are well known under the local names Aleppo evil, Biskra button, Button de Crete, Delhi boil or sore, as also Garzebad, Jeypore, Lahore, Moultan, Meerut, Roorkee, Scinde, Umballa, Agra, and Aden boils or sores. Until very recently authorities differed as to the identity of these boils, but now almost all agree, and, for practical purposes at any rate, they may be said to be identical.

All ranks, sexes, ages, and classes of the population may be affected, and the exposed parts of the body are very prone to attack, although the boils may appear all over the body, except upon the palms of the hand and soles of the feet, and they are rare upon the scalp. Children are usually attacked on the face.

These boils are most frequently met with during the rainy season, and vary in number from one or two to fifteen or even more, although the smaller number is the most usual. Horses and dogs probably also suffer from this affection.

Debility certainly predisposes to boils, as also the combined effects of malarious influences, exposure to long-continued heat and residence in insanitary localities, aided perhaps occasionally by a scorbutic or syphilitic taint.

The *etiology* of this affection is in an unsatisfactory state. Lewis and Cunninghame thought that there was no evidence of any parasitic agency in the production of the disease, and considered that it was due to the chemical constituents of the water. Vandyke Carter considered that Delhi sore and Button de Biskra were identical, and thought it due to some fungus. Other observers think that the disease is due to inoculation by some insect.

If the boil be examined microscopically, infiltration of the skin and the areolar tissue, with lymphoid and epithelioid cells, is seen; these cells cluster round the blood-vessels and lymphatics. The nuclei of the cells are from 3 μ to 6 μ, the cells themselves from 7 μ to 9 μ. In 1885 Cunningham made a further investigation, and said that the diseased processes might be associated with and possibly caused by peculiar parasitic bodies which he found; they could be stained by gentian violet. His researches were confirmed by Frith, but Riehl subsequently found micrococci, as did Poncet, varying in size from 0·9 μ to 1·0 μ in the granulation cells. The whole matter is, however, at present indefinite.

The incubation of the disease varies from 3 to 16 days. After one attack a certain local immunity is enjoyed, and Bagdad Jews are said to have inoculated their children so as to prevent their disfigurement. The value of this practice is however doubtful, as the affection can be undoubtedly produced by inoculations upon persons who have previously suffered from it, but here again information is not absolutely accurate, and local rather than general immunity is most probable.

The *symptoms* of this affection are first itching, followed by the appearance of a reddish spot, which usually surrounds a hair follicle. A papule results, which gradually enlarges until a more or less appreciable tubercle, of a shotty feel, results. This in a few days softens, breaks down, and an ulcer is developed, which ulcer gradually spreads until an area of some two or three inches in diameter may result in severe cases. It is far more usually limited to an ulcer of perhaps an inch in diameter. The ulceration spreads by erosion; the margins are perpendicular, ragged, and surrounded by an areola. The surface of the ulcer discharges a sero-purulent matter which forms scabs, as a rule covering the entire lesion. The onset of the disease is accompanied by more or less constitutional disturbance, malaise and fever being usually present and sometimes severe. The duration of the affection varies; sometimes the ulcer is small and heals rapidly, but it may last for a year. The duration is not much influenced by treatment in the present state of our knowledge.

With regard to *treatment*, great cleanliness is necessary to avoid auto-infection. The constitutional condition must be carefully inquired into, and tonics, such as iron, quinine, arsenic, and the mineral acids, are indicated. Change of air is very beneficial. In severe cases the patients should be sent either home or to some local sanatorium. The sufferers should be well supplied with nourishing food; stimulants may be required, and the bowels should be carefully regulated. Local treatment must depend upon circumstances. If the case is seen early, Dr. Fleming's treatment of applying strong mineral acid or potassa fusa to the pimple several times, so as to thoroughly destroy it and turn it into an ulcer, may be recommended, the ulcer being treated on general principles subsequently, but not irritated. Native practitioners often use the actual cautery in the early stage, and the writer has found this useful, as also Volkmann's spoon. If the sores are small in extent they may be covered by iodoform collodion. If the pain is severe, as it sometimes is, belladonna ointment is indicated, or a compress of a 10 per cent solution of chloral hydrate in glycerine and water. It is well to remember that after healing these boils frequently leave a scar, which, if on the face, may be unsightly. Patients should be warned of this; sometimes a scar may take the form of a brown mark; hence the name "date mark" given in some regions to the affection.

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Gall-Bladder and Bile Ducts, Diseases of.

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Anatomy.—The gall-bladder is a pear-shaped sac, measuring three and a half inches in length and two inches at its greatest breadth, which lies against the under surface of the right lobe of the liver, filling up a slight fossa to the right of the quadrate lobe and projecting into the peritoneal cavity.

Its fundus, when the gall-bladder is distended, normally projects slightly beyond the free margin of the liver immediately under the ninth costal cartilage, and from this position the cyst passes backwards and slightly to the left, gradually narrowing until its neck ends in a sigmoid curve, the terminal portion of which turns downwards to its junction with the cystic duct near the right extremity of the transverse fissure of the liver. Its upper surface, as a rule, is attached to the under surface of the liver by connective tissue, the peritoneum covering the fundus and lower surface, and passing thence to the liver; but occasionally it is completely invested by peritoneum. At times also a mesentery is formed on the under surface by an extension outwards of the free border of the lesser omentum to the fundus.

The cystic duct passes backward, slightly downward, and to the left for rather less than one and a half inches, and joins the hepatic duct at an acute angle to form the common bile duct. The hepatic duct measures about two inches in length, and is formed by the junction at a very obtuse angle of a branch from the left, and another from the right lobe of the liver, which issue at the transverse fissure. Thence it descends somewhat to the right within the gastro-hepatic omentum, lying in front of the portal vein, and having the hepatic artery to its left.

The common duct averages three inches in length and passes backward and downward between the layers of the gastro-hepatic omentum, having the same relation to the portal vein and hepatic artery as the hepatic duct. Passing behind the first part of the duodenum it continues downward on the inner and posterior surface of the second part in intimate relation with the head of the pancreas, and for a short distance in contact with the right side of the duct of Wirsung. Along with this duct it perforates the muscular coat of the duodenum, runs obliquely for three-quarters of an inch in the submucosa, and they open into the lumen of the bowel by a common orifice three inches from the pylorus.

The cystic duct normally admits a No. 5, the common duct a No. 7 catheter; but at different parts they both vary in size, the cystic being narrowest at its termination, while the common duct shows two dilatations, one just at its beginning and another near its termination. At its entrance into the duodenum it is so narrow that it only admits a fine probe.

The ducts have each three coats,—serous, muscular, and mucous,—the last being studded with mucus-secreting glands. In the cystic duct the mucous membrane is thrown into folds, which, especially at its termination, act somewhat as a valve.

The gall-bladder, except where in contact with the liver, has the same three coats, and its mucous membrane also is abundantly supplied with glands which secrete mucus. Its normal capacity is about six drachms, but under varying pathological conditions its size undergoes great alterations.

The gall-bladder gets a free supply of blood from a branch of the hepatic artery—the cystic artery—which in turn sends a branch along the cystic duct to anastomose with an offset from the gastro-duodenal artery. The veins empty themselves into the portal vein, and the lymphatics drain into glands which lie in the free border of the lesser omentum.

Accompanying the arteries are branches from the cœliac plexus of the sympathetic.

The lower surface of the gall-bladder is in relation with the hepatic flexure of the colon, and, toward its neck, with the first part of the duodenum and occasionally the pylorus. The tip of the fundus just touches the anterior abdominal wall, and occasionally the right border of the great omentum lies in contact with it. In pathological conditions, however, there may be great alterations in the relations of the parts.

Congenital Malformations.—The gall-bladder is sometimes partly bifid, or by a central constriction it may assume an hour-glass shape. Occasionally it and even the larger bile ducts are absent or represented merely by fibrous cords.

A malformation of the liver not infrequently met with is of great importance in gall-bladder work. It consists of a tongue-shaped prolongation of the right lobe, which may project below the costal margin for several inches and carry the gall-bladder down with it, though it is more common for the cyst to be situated just to the left of the abnormal (Riedel's) lobe.

Congenital Obliteration of the Bile Ducts is sufficiently common to merit special attention. The exact cause of this condition has not been determined, but it apparently consists of an inflammatory process affecting the bile ducts during intra-uterine life, tending to be followed by complete obliteration of their lumen and associated with some degree of cirrhosis of the liver.

It is probable that the cases which have been reported from time to time under the title "absence of the gall-bladder and bile ducts" should really be relegated to this category, as it is not unusual for some part of the extra-hepatic biliary apparatus to be absent in this disease. Where the patient has lived some time it is usual to find more or less marked biliary cirrhosis, and the liver occasionally, and the spleen usually, are somewhat enlarged.

In a few cases there has been reason to suspect the presence of congenital syphilis; but in the majority the parents have apparently been in perfect health.

The male sex suffers more frequently than the female, and in several recorded instances several members of a family have been affected by the disease.

That the process is a local inflammatory disturbance is evident from the fact that at birth the patient has usually been of normal weight, and seemed normal in all respects.

As a rule the first symptom pointing to any serious condition is the progressive deepening of the jaundice which is either present at birth or soon supervenes. An early symptom in the disease is the occurrence of hæmorrhages from various parts—from the umbilicus after separation of the cord, from the nose, stomach, or intestine, or into the subcutaneous tissues. Where the affection has started early in intra-uterine life the fœtus will from birth be pale, but if toward the end of the period of gestation, normal meconium will first be extruded, and this will be followed by clay-coloured stools. Unless death results from hæmorrhage, the general nutrition suffers considerably after a time; but, if not carried off by some intercurrent disease, the patient may live on for months before succumbing to the effects of the continued malnutrition.

The only condition with which this disease is likely to be confounded is icterus neonatorum, and in the early stages it is impossible to distinguish between them; but the continuance of the jaundice and the supervention of serious sequelæ soon render a diagnosis easy.

The prognosis is uniformly bad; even in those cases where there is merely narrowing of the ducts and inspissation of the bile, life has never been prolonged more than eight months.

So far no treatment has seemed to do any good; but since the disease consists in a local inflammation it will be well that the duodenum should be kept as aseptic as possible by the administration of gray powder, and that the bile be kept as thin as possible by giving as much fluid as can comfortably be ingested, in the hope that if the disease has not progressed to obliteration of the ducts the process may stop.

INJURIES OF THE GALL-BLADDER OR BILE DUCTS are not common, but several have been reported. The symptoms vary considerably according to the condition of the bile, which may or may not be pathological. Practically invariably in cholelithiasis, or in any inflammatory condition affecting the bile channels, the contents of the gall-bladder and ducts contain pyogenic microbes, and if, in the presence of these, the ducts or gall-bladder are so injured as to lead to extravasation, an acute septic peritonitis is set up, which even if promptly treated is very apt to end fatally, though recoveries have been reported. Where, however, the bile is normal, no micro-organisms are present, and, even if extravasation takes place, there is less risk of immediate septic peritonitis; though, if the extravasated fluid be not removed, peritonitis will certainly supervene sooner or later.

Many of the recorded cases of rupture of the gall-bladder have occurred from comparatively slight sudden pressure, induced by straining at stool, by vomiting, by sneezing, or by blows over the hepatic region; but in these cases, in all probability, there has been predisposition to rupture in the shape of thinning from ulceration or long-continued distension. The course and symptoms in such a case will be those of perforation of the gall-bladder, a subject which will be considered below.

In non-pathological conditions of the gall-bladder and bile ducts the injuries, in the reported cases, have taken the shape of gunshot wounds, of perforating abdominal wounds by sharp instruments, or of the passage of a heavy conveyance over the hepatic area. In cases where a perforating abdominal wound is present operation would probably be done as soon as possible, and before any symptoms pointing directly to injury to the bile channels had arisen. On the true nature of the condition being determined the extravasated bile should be mopped out by sponges, the rent in the viscus closed by Lembert sutures, and drainage of the right kidney pouch established. If the patient does not die from the shock of the injury, and operation is undertaken sufficiently early, the chances of recovery from such a condition should be very good.

Where the injury has resulted from the passage of a heavy body over the abdomen, there has not in uncomplicated cases been much evidence at first of any serious visceral lesion.

In a few days after the accident jaundice will appear along with symptoms of peritonitis, which may be general, but is not at the outset very acute, though it ultimately becomes so. Examination of the abdomen will give evidence of the presence of free fluid in the peritoneal cavity, and the stools will be clay-coloured or paler than normal.

Whenever it is clear that there has been escape of bile into the peritoneal cavity the abdomen should be opened, the free fluid mopped out by sponges if localised, or flushed out by sterile normal salt solution if free in the general cavity of the peritoneum, the wound closed if possible, and drainage established.

If this course is pursued the prognosis is good, one case being reported in which recovery took place, although the operation was not undertaken until apparently thirty days after the injury to the bile channels.

CHOLELITHIASIS

Though gall-stones are extremely frequent, various estimates giving them as occurring in from 4 to 12 per cent of all autopsies, yet it is only in a minority that any distinct clinical evidence of their presence is afforded.

In number and size the gall-stones found vary greatly in different cases. Occasionally a single stone is found, while as many as 7802 have been obtained in one case; they may be so small as just to be perceptible, while, on the other hand, one has been described 5 inches by 1¾ in.

As a rule a single stone is rounded or pyriform, but when many are present they are more usually small and faceted. In colour they vary from almost pure white to dark olive green, according to the relative amount of bile pigment present. As might be supposed from their being composed almost entirely of cholesterin, they are at times sufficiently soft to be readily crushed between the finger and thumb, but on the other hand they may be firm and hard. Their specific gravity is just above that of water.

The cholesterin, which is the main constituent in a biliary calculus, is derived, not as used to be thought from the bile, but from the epithelial lining of the gall-bladder or bile ducts; indeed, the excretion from mucous membranes generally, especially if inflamed, contains quite as large a proportion of cholesterin as does the bile.

It seems probable, therefore, that the origin of gall-stones is to be sought, not in any alteration in the characters or rate of flow of the bile, but rather in a local condition of the mucous membrane of the bile passages. During the last twenty years from all sides evidence has been accumulating which tends to show that this preliminary condition consists of a bacterial infection of the bile channels from the intestine. Normally bile is sterile, but in practically all cases of cholelithiasis micro-organisms can be obtained if carefully searched for. As a rule the bacillus coli communis is the microbe found, but the typhoid bacillus, staphylococci, and streptococci have also been found, and it is probable that other bacteria may induce a cholangitis sufficient to give rise to a hypersecretion of cholesterin.

More recently gall-stones have actually been experimentally produced by

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inserting into the gall-bladder various species of bacteria specially attenuated by cultivation in media containing increasing proportions of bile. It may, therefore, for practical purposes, be taken as proved that gall-stones owe their origin to a bacterial invasion of the bile channels, giving rise to a catarrh of the mucous membrane and a deposition of that portion of the cholesterin secreted which is in excess of what the bile salts are able to hold in solution. The mere presence of this deposit will, of course, keep up the catarrh even should no further microbic infection take place.

While this is so it is necessary that we should consider several conditions which seem to predispose to cholelithiasis.

Of these the most important is the character of the food. There seems to be little doubt that the ingestion of excess of carbohydrates as compared with nitrogenous food is a fairly common precursor of the onset of gall-stones. While this may be due to the fact that, with a diminution in the amount of albuminous material consumed, there is a corresponding deficiency in the bile salts which dissolve cholesterin, it seems more probable that it is associated with an increased tendency to catarrh of the stomach and duodenum from a greater liability to abnormal fermentation in the stomach. Normal gastric juice seems to kill most disease-producing bacteria, and it has seemed to us probable that any alteration of the contents of the stomach and duodenum would tend to increase the risk of the introduction of microbes from the bowel into the common duct.

The taking of alcohol, except in moderation, seems also to predispose to cholelithiasis, and this may act in a similar fashion.

Gall-stones may occur at any age from infancy upward, but they are distinctly more prevalent in the later decades of life. Why this should be so is not on the surface quite apparent; but it has been suggested that the reason for biliary calculi being found more frequently in autopsies on elderly subjects is that the walls of the bile passages undergo great atrophy in old age, and are consequently unable to expel any small concretions which form.

Sedentary habits, from whatever cause arising, appear undoubtedly to predispose to cholelithiasis. Lack of sufficient exercise, besides affecting prejudicially the general tone of the tissues, is not infrequently associated with dyspepsia and gastro-intestinal disturbance, and it is possible this is the explanation of the coincidence. It is probable, also, that the defective respiratory movement and absence of contraction of the abdominal muscles tend to help toward more or less stagnation of the bile in the gall-bladder and bile passages. It has been found that if the bacillus coli be injected into the gall-bladder of a dog after the common duct has been ligatured, an acute cholecystitis develops, whereas if the ducts are free no bad symptoms appear. Similarly the stagnation of bile consequent on deficient exercise may tend toward the more ready origin and propagation of a catarrh of the ducts.

Gall-stones occur much more frequently in women than in men—in the proportion of about 3·5 to 1. This is probably associated with their taking less exercise, but it is possible that the habit of wearing corsets, which depress the fundus of the gall-bladder, and so impede the flow of bile from it, helps as an etiological factor.

Symptoms.—The symptoms caused by gall-stones vary greatly in different cases. It is probable that this is in the main due to the locality in which they are situated and to the presence or absence of complications.

In this section only uncomplicated cholelithiasis will be considered, the complications being relegated to a later chapter—a method which, though pathologically somewhat illogical, is clinically convenient.

(*a*) *Gall-stones in the Gall-bladder.*—Though most frequently found in the gall-bladder, it is a mistake to suppose that all biliary calculi originate there. So long as they remain in the gall-bladder it is probable that they give rise to no special symptoms, unless possibly some sense of slight uneasiness and discomfort in the epigastrium, and some tenderness below the right costal margin, if the inflammatory condition, which gave rise to their formation and which is perpetuated by their presence, has spread to the peritoneal covering of the cyst.

(*b*) *Gall-stones in the Cystic Duct.*—When a calculus reaches the cystic duct there is sooner or later some symptom of its presence. A typical attack of gall-stone colic, when the stone is lodged in or extruded into the cystic duct, consists of very severe pain beginning under the right costal margin, and radiating thence toward the epigastrium and umbilicus, and round the right side, usually toward the subscapular region, but occasionally even to the right shoulder. The onset of pain is often apparently without any determining factor, though in some patients it will be found usually to occur after taking food. Its duration varies very considerably in different patients, and at different times in the same patient. Not infrequently it ends with the onset of vomiting. In a severe seizure there is very great collapse, so that the condition may closely simulate angina pectoris, perforation of a gastric ulcer, or other grave abdominal catastrophe, and death has on more than one occasion resulted from the severity of the pain.

The vomiting may persist after the pain has quite ceased, and the stomach may continue irritable for days after, so that the ingestion even of liquid food brings on another attack of vomiting, and resort has to be had to rectal alimentation. If the common duct is free, some bile will usually be ejected after the contents of the stomach have come away.

Associated with this seizure there is a greater or less degree of local peritonitis giving rise to rigidity of the upper segment of the right rectus muscle and tenderness over the gall-bladder region, particularly at a point about midway between the ninth costal cartilage and the umbilicus.

During such an attack there is frequently some distension of the gall-bladder, but this can rarely be made out on account of the muscular rigidity and the excessive tenderness.

Jaundice is not usually an accompaniment of gall-stone colic in these cases, though it may appear at the end of twenty-four hours and persist as slight icterus for two or three days. In such a case it is due to catarrh spreading to the common or hepatic duct, and interfering with the flow of bile. It is not common to find a stone in the motions after such an attack as that described above, but one or more may be found.

It is important to remember that neither the absence of calculi nor a failure in the appearance of jaundice negatives the diagnosis of cholelithiasis.

While the description given above applies to a typical cholelithic attack it must not be supposed that in every case the symptoms will be so definite or so severe; but even the milder seizures, so-called "spasms," will be found to conform more or less, and to differ only in the intensity of the individual symptoms.

They may be repeated at intervals of a few days, or months may intervene between one attack of colic and another. In the intervals the patient may be quite comfortable, but local examination will usually detect a point between the costal margin and the umbilicus where deep pressure will elicit tenderness, and if a gall-stone be impacted in the cystic duct a tumour may be felt, due to the gall-bladder being distended with mucus.

(*c*) *Gall-stone in the Common Duct.*—The symptoms of gall-stone colic, when the calculus is in the common duct, resemble in many ways those following on the impaction of a stone in the cystic duct, but differ in respect to the seat and distribution of pain and to the occurrence of jaundice.

The pain in such a case begins in the epigastrium, and radiates more to the left side of the abdomen and through to the inter-scapular space. The tenderness will also be found most marked in the middle line about midway between the umbilicus and ensiform cartilage.

Jaundice is invariably present, but varies in intensity according as the stone does or does not completely block the duct, or become impacted at its entrance into the duodenum. Where the stone is too small quite to fill the lumen of the tube the jaundice will resemble that found in the last class, but its onset will be earlier, and it will last longer. Here, as before, the immediate cause of the icterus is the inflammatory swelling of the mucous membrane, and not the stone itself. Should the stone quite fill the duct, or should it become impacted at the entrance to the bowel, the jaundice will be persistent, and may become extreme. In that event there will be much greater chance of rapid deterioration of health, and greater difficulty in diagnosing the condition from malignant disease. There will also be the usual signs and symptoms of deep jaundice, especially, what from a surgical point of view is of great importance, a marked tendency to hæmorrhage.

As was found to be the case when a stone was present in the cystic duct, so here, between the seizures, if the jaundice is not persistent, the patient may be fairly well, though interference with the general health is much more frequent than in the former case. Tenderness will always, or nearly always, be elicited if deep pressure be made midway between the umbilicus and the tip of the sternum.

In many cases infective cholangitis supervenes, a condition which will be described later.

Diagnosis.—In uncomplicated cholelithiasis the diagnosis will not as a rule present much difficulty. The situation, character, and distribution of the pain and tenderness along with the accompanying symptoms will usually be sufficient to render the case clear. The locality in which the stone is situated can, for the most part, be determined by noting the point of greatest tenderness. When this is in the middle line the calculus will probably be in the common duct, and the pain in the back will be more central; whereas if the stone is in the cystic duct the point of greatest tenderness will be somewhere along a line between the umbilicus and the ninth right costal cartilage, and the pain will be felt to radiate to the right subscapular region. Should the pylorus, however, be adherent to the cystic duct at the site of the stone the pain and tenderness may simulate closely that caused by a calculus in the common bile duct; but in such a case there will usually be some degree of dilatation of the stomach or other gastric symptoms pointing to this complication.

If the common duct is quite blocked, jaundice will become intense, and there may be great difficulty in eliminating malignant disease as a probable cause of the illness. The previous history of attacks of "spasms," and particularly the account of severe pain at the onset of the illness, together with local tenderness, will be important indications tending to show that the disease is simple. On the other hand, in malignant disease of the head of the pancreas or in the bile ducts, severe pain is not a common symptom, though it does occur, and the wasting and deterioration of the general health are more rapid than in jaundice due to impacted stone. Where the cause of the disease is simple it is distinctly unusual to find a distended gall-bladder; but in obstructive jaundice dependent on malignant disease this condition is the rule. It should, however, be remembered that gall-stones are probably invariably present before primary malignant disease of the gall-bladder or bile ducts, and in any particular case both conditions may occur. The absence of severe pain at the outset of the last attack of persistent jaundice in such a case may be so striking as to lead the patient himself to remark on the fact, as was the case with a gentleman from New Zealand whom we saw recently suffering in this way.

Should diagnosis be impossible, an exploratory incision will clear up matters, or it may be advisable to wait to see whether secondary nodules appear in the liver.

Aspiration of a distended gall-bladder or sounding for gall-stones should never be resorted to, as neither gives any information which cannot be obtained by a careful examination of the patient, and both operations are dangerous.

Treatment.—Once gall-stones have formed, it is probable that no means at present at our disposal suffice to dissipate them; but as some medical authorities hold a contrary opinion it will be well to consider here the various remedies which are supposed to have this power.

The drug which is most in favour at the present time is olive oil. It should be given in large quantity, as much as half a pint per day being recommended by some. There can be no doubt that if the oil could reach the calculi in the gall-bladder or bile ducts, and remain in contact with them, it would bring them into solution, as olive oil has been found to dissolve 68 per cent of a gall-stone placed in it for two days. Oleic acid has a similar but rather more rapid effect, and a solution of animal fat also has a tendency to soften biliary concretions. There is, however, no evidence to show that ingested oil can come into direct contact with calculi in the bile passages. It has been suggested that the good effect said to be obtained may be explicable on the supposition that an increased absorption of fat in the form of fatty acids and soaps leads to a greater proportion of these in the bile, and that they are the active ingredients in causing the dissipation of the concretions.

It seems, therefore, just possible that the administration of oil may in some cases, especially where the concretions are small, lead to their dissolution, or so reduce their bulk as to permit them to pass; but it should be remembered that excess of fat in any form is apt to induce dyspepsia and catarrh of the stomach and duodenum, and in this way may tend to aggravate the condition which it is sought to relieve.

So far as our own experience goes, we cannot say that in any case, even after treatment by this means continued over a lengthened period, we have seen any such material benefit as other writers describe.

The so-called saline cholagogues do not affect gall-stones introduced into solutions containing them, so that their administration is not likely to have much effect in ridding a patient of calculi once they are formed, though they may relieve the associated catarrh.

Choloroform, ether, turpentine, and several other substances readily dissolve cholesterin; but there is no evidence to show that, when taken by the mouth, they are excreted in the bile in such quantity as to have any effect in removing gall-stones in the gall-bladder or bile ducts.

During an attack of cholelithic colic the pain is so severe that not infrequently it is necessary to give morphine; but in other cases, where the pain is less intense, it may be relieved by the administration of a tumblerful of water as hot as it can comfortably be taken and the local application of hot compresses.

Surgical Treatment.—(*a*) When gall-stones in the gall-bladder or cystic duct give rise to symptoms, though urgent need for operative interference is less than in those cases in which the common duct is occupied by a calculus, yet in a certain number operation will be called for, notwithstanding the absence of those complications, inflammatory and other, which will be discussed later.

In simple cholelithiasis the two conditions which would seem to indicate necessity for surgical interference are gall-stone colic, recurring so frequently as to interfere with the general health of the patient or prevent him filling his position in society, and enlargement of the gall-bladder without jaundice, even in the absence of pain.

It should also be remembered that delay in resorting to operation means increased risk, from shrinkage of the gall-bladder and the formation of adhesions, should it become necessary to operate later; while there seems little doubt that the continuous irritation from the presence of gall-stones predisposes to malignant disease of the gall-bladder and cystic duct. In addition there is always the risk of acute inflammatory disturbance supervening, and operation having to be done with the patient in a bad condition to withstand surgical interference.

Cholecystotomy.—In such a case as we are considering, the operation which will probably be necessary is that of cholecystotomy. A vertical incision through the outer border of the right rectus is that which we prefer, but some surgeons habitually use an incision through the right semilunar line, while Kocher has advised, and many surgeons employ, an oblique incision parallel to the right costal margin. If, on opening the abdomen, the gall-bladder be found distended, it should be aspirated, and then opened, after the parts have been isolated by flat sponges. A pair of forceps or a small scoop should then be introduced, and any gall-stones present in the gall-bladder removed. After the gall-bladder has been cleared, the fingers are passed along the outside of the ducts (any adhesions which may have been present having previously been broken down), and the cystic duct is searched for calculi. If any be found they should be manipulated backward into the gall-bladder and thence removed. Sometimes it is necessary to crush a stone in the duct before it is possible to return it into the gall-bladder; at other times the calculus is so large or so hard that this manœuvre is impossible, and then it becomes necessary to incise the duct in order to remove the concretion. Should this latter course be adopted, the incision in the duct should be carefully stitched up, a continuous catgut suture being used for the mucous membrane, and a continuous silk suture for the peritoneal investment.

The gall-bladder and cystic duct having thus been cleared, a non-perforated rubber tube should be inserted into the opening in the gall-bladder, and the edges of the incision brought up and sutured, by fine chronic catgut, to the abdominal wall, the serous coat being stitched to the parietal peritoneum and the mucous membrane to the aponeurosis. The drainage-tube may be shortened on the second or third day, and removed a few days later; but, if there has been much cholecystitis, drainage should be maintained for a longer period.

When it is not possible to bring the gall-bladder up to the anterior abdominal wall the tube should be inserted into it, and a purse-string suture applied round the margin of the incision in the gall-bladder, so that when tightened it will draw the edges of the incision closely round the tube. This plan is, we consider, much better than that suggested by some surgeons, of stitching up the incision in the gall-bladder, since it seems to us of the utmost importance that, if recurrence of cholelithiasis is to be prevented, the catarrhal condition of the mucous membrane should be removed by the employment of free drainage.

(*b*) When the gall-stones are in the common duct, operation is imperative; but the particular measure to be adopted is not always clear, and will vary in different cases.

Choledochotomy.—The operation of election is choledochotomy, but at times the condition of the patient is such as to render the choice of this out of the question by reason of its difficulty and danger. The duct may be opened and the stone extracted, either by direct incision through the wall of the canal at the site of the calculus, or by incising the second part of the duodenum and stretching the papilla, or slitting up that part of the duct which runs in the posterior wall of the duodenum. The duct, or bowel, should then be carefully sutured, and the gall-bladder may or may not be drained in the manner already described. Separate drainage of the peritoneal cavity will also as a rule be advisable.

Should the condition of the patient render this operation too dangerous, the stone may at times be crushed in the duct between the finger and thumb, in the hope that the fragments will pass on into the intestine, or that they may be dissolved by repeatedly injecting through a drainage-tube, introduced into the gall-bladder, olive oil or a 0·5 per cent solution of sapo animalis.

Cholecystenterostomy.—In a few cases it may be advisable to perform cholecystenterostomy, *i.e.* to make a direct artificial communication between the gall-bladder and duodenum. When the gall-bladder is distended, this is a very simple matter; but as a rule, in these cases, the gall-bladder is contracted, and the operation then is one of great difficulty and very considerable danger, scarcely, if at all, less severe than choledochotomy. In our experience the junction has been most expeditiously effected by the aid of a Murphy button.

In all cases of cholelithiasis, after recovery from operation, the patient should be instructed to attend to certain details of after-treatment. The bowels should be kept regular, preferably by the use of mild salines, of which probably the natural Carlsbad water is the best. The diet should be so regulated as to obviate the likelihood of dyspepsia, and, for this reason, the patient should be advised to avoid over-indulgence in sweet or starchy foods, or highly seasoned dishes. A sufficiency of albuminous food should be taken, either in the shape of meat or fish, or of vegetable food-stuffs which contain a large proportion of nitrogen. Alcohol should be avoided except at meals, and then should be well diluted.

A sufficient amount of exercise should be taken daily, as thereby the general nutrition will be improved, and the likelihood of fermentative changes taking place in the contents of the stomach and duodenum diminished.

CHOLECYSTITIS

Simple catarrh of the gall-bladder, like that of the bile ducts, may be either acute or chronic. As the acute affection is always associated with a similar condition in the ducts, and as it is the latter that gives rise to the most evident symptoms, the consideration of the former will be deferred until the latter is discussed.

A chronic catarrh, as has already been pointed out, is probably an invariable precursor of the formation of gall-stone in the gall-bladder, but, as a rule, this condition gives rise to few symptoms other than evidences of dyspepsia, until calculi have formed.

The form of chronic catarrh which does give rise to symptoms is that usually following on the presence of gall-stones and persisting after these have disappeared, but occasionally originating as the sequel to an acute cholecystitis, like that sometimes present in association with typhoid fever, influenza, and other acute affections.

In these cases the local condition depends in great measure on the duration of symptoms. Where these have lasted only for a short time the gall-bladder may be somewhat dilated, though it is rare to find it so large as to be palpable through the abdominal wall; on the other hand, if the patient has been suffering for a long period the walls of the gall-bladder tend to become thickened, and the gall-bladder itself tends to contract. Adhesions to the neighbouring viscera may or may not be present according as the inflammatory disturbance has or has not extended to the peritoneal coat. As a rule, however, if there has been well-marked and repeated gall-stone colic, or if the preceding cholecystitis has been acute and associated with marked tenderness, there will be more or less adhesion present. The gall-bladder will usually be found to contain thick, ropy mucus, sometimes so inspissated as to resemble grains of boiled sago; but it may be so contracted that the cavity is practically obliterated,—a form to which the term *cholecystitis obliterans* may be properly applied.

The symptoms in this condition simulate very closely those due to cholelithiasis, but the colic is for the most part less severe, and the tenderness is either absent or much less marked. Jaundice is almost invariably absent, but, rarely, it does occur. In one case we treated recently the attacks of colic were invariably preceded by slight jaundice; in this case it seemed as if the thickened mucus was allowed to pass until a catarrhal condition of the mucous membrane in the common duct so lessened the calibre of the passage as to retard the flow of the mucus and thus set up painful spasm.

Medical treatment similar to that advised for acute catarrh will, if persisted with, get rid of symptoms in most cases; but in some, especially where there are adhesions, the gall-bladder will require to be opened and drained until the inspissated mucus disappears from the discharge. At the same time any adhesions which are present should be broken down. If the cavity of the gall-bladder be almost or altogether obliterated, in all probability the best mode of treatment will be to perform cholecystectomy.

Empyema of the Gall-bladder.—Normally bile removed from the gall-bladder will be found sterile, but experimentally it has been shown that if the outflow of bile and mucus from the gall-bladder be obstructed pyogenic micro-organisms make their appearance.

Pure cultures of staphylococci, streptococci, and bacillus coli communis have been introduced into the gall-bladder, where there was no obstruction to the outflow, without producing any untoward symptom. This experimental evidence is supported by clinical experience, since in every case in which empyema of the gall-bladder occurs it will be found that there has been present some cause of obstruction in the cystic duct; either a gall-stone has become impacted in the duct, or the lumen has become diminished as a result of a chronic catarrh, of kinking from adhesions, or of malignant disease.

In the great majority of cases the onset of empyema is preceded by cholelithiasis, though it may occur as a consequence of obstruction of the cystic duct from any cause—chronic catarrh, cancer of the duct, hydatids, etc.

From whatever cause arising, the onset of suppuration is followed by dilatation of the gall-bladder and localised peritonitis in its neighbourhood. Should the case be allowed to progress, the gall-bladder will either rupture into the general peritoneal cavity, giving rise to peritonitis, or it will contract adhesions to the neighbouring hollow viscera or the parietes, and relief may occur by the discharge of pus either into the intestinal canal or through the skin.

The symptoms leading up to empyema will, of course, vary with the cause, but mostly there will be the ordinary symptoms of cholelithiasis. With the supervention of suppuration there will appear a swelling under the right costal margin. The tumour will be found to be somewhat pear-shaped, and will be directed along a line reaching from the tip of the ninth costal cartilage to a point in the middle line about an inch below the umbilicus. It will move with respiration in the earlier stages, and will be only slightly tender. As the inflammation spreads to the peritoneum the tenderness will become greater, and the outline of the tumour will become less distinct—partly from the adhesions which it contracts, but mainly from the muscular rigidity which now appears. Pain will be continuous, but it varies in severity. In some cases there may be no elevation of temperature, but in others even at the commencement there are fever and malaise, and rigors may occur. In our experience the presence of fever has always been found to be associated with ulceration of the mucous membrane of the gall-bladder, and the pain has been more severe than in those cases where the mucous membrane was intact. Where there are no constitutional symptoms the general condition of the patient may not deteriorate much, but if fever and great pain be present he will lose strength and weight.

Immediately the diagnosis of empyema of the gall-bladder has been made, cholecystotomy should be performed, and drainage of the abscess cavity must be continued till the discharge is sterile. If the condition of the patient is such as to permit of it, the cause of the obstruction in the duct should be removed; but, in some cases where operative interference has been delayed, it may be well to content one's self with drainage of the cyst, and to leave the removal of the cause to a later date.

Phlegmonous cholecystitis is induced by a much more acute infection of the gall-bladder than that which gives rise to empyema, and consequently runs a much more rapid course, usually terminating fatally in a few days; though, if early operative treatment were in all cases adopted, there does not seem to be any reason why it should have a higher rate of mortality than has acute appendicitis. The gravity of the condition seems to depend on the fact that infective peritonitis is set up very rapidly before any localising adhesions have had time to form, the microbic contamination apparently arising without any direct gross communication between the interior of the gall-bladder and the peritoneum.

In these cases the walls of the gall-bladder become rapidly swollen, soft, and œdematous, the peritoneal coat loses its lustre, and there rapidly supervenes an acute peritonitis originating in the right upper part of the abdomen, leading to paresis of the intestinal coils located in that region, and giving rise to symptoms of intestinal obstruction. The gall-bladder will be more or less distended with muco-pus, and its surface will be purplish or even have a green tinge. If the patient survive, and nothing be done to relieve him, the gall-bladder will necrose in patches, but as a rule death takes place from septic peritonitis before this stage is reached. If death does not rapidly ensue, adhesions may form, limit the inflammatory disturbance, and lead to the formation of a localised abscess, which will follow a course similar to that of the abscess due to acute appendicitis.

Generally acute infective cholecystitis is associated with cholelithiasis, but in one recorded case it appears to have followed on cholecystitis occurring during an attack of typhoid fever.

Symptoms.—Either as a sequel to a prolonged history of "spasms," or apparently spontaneously, the patient is seized with sudden acute pain in the right hypochondrium radiating to the epigastrium, and through to the right subscapular region, and which rapidly spreads till the whole abdomen becomes affected. Associated with this are the usual signs at first of local, later of general peritonitis. In the earlier stages there are tenderness and some distension below the right costal margin, with rigidity of the right rectus, but soon there comes general abdominal tenderness and tympanites, though even in the later stages the tenderness is most intense in the right hypochondrium. As occurs in all cases of acute peritonitis, there are a rapid thready pulse, quick thoracic breathing, and more or less collapse.

Before rupture has taken place the temperature will usually be found elevated, but when general infection has occurred the temperature curve may give little indication of the patient's condition. As a rule there will be no jaundice, as the disease progresses too rapidly to permit of its appearance, but in the more chronic cases it may be present.

Unless there is a previous history of gall-stone colic the diagnosis of the exact condition in phlegmonous cholecystitis is difficult, and usually it will only be possible to say there is acute peritonitis beginning in the right side of the abdomen. The site of the original pain and the direction in which it radiates, together with the greater tenderness in the right hypochondrium, and the primary appearance of distension in that region, may help one to come to a decision. Fortunately the conditions which it simulates—fulminating appendicitis and perforation of some hollow viscus—demand the same initial treatment, viz. exploratory laparotomy.

Treatment.—At the onset the pain is so severe that a hypodermic injection of morphine will always be necessary. If the tenderness be not too great, local hot applications may be used, and all feeding by the mouth should be stopped.

Whenever there is a fair presumption that the case is one of phlegmonous cholecystitis, or, in the event of no accurate diagnosis being possible, it is found that the patient is gradually getting worse, and the evidence of general peritonitis becomes more manifest, the abdomen should be opened. If gangrene has supervened, cholecystectomy should be performed; but if the disease has not progressed so far, it will be sufficient to aspirate the contents of the gall-bladder, then to open it and drain. For the most part the condition of the patient will be such that unless this can be easily accomplished it will not be wise to make any attempt to remove the cause at the first operation. Should there be much peritonitis it may be wise at the same time to drain the right kidney pouch, either by gauze introduced through the wound in the anterior abdominal wall, or by making a counter-opening in the loin. In subacute cases, where an abscess has formed between adhesions, this should be drained, and when the patient's condition is better cholecystotomy can be done.

Croupous Inflammation of the Gall-Bladder and Bile Ducts.—Recently renewed attention has been directed to a form of cholecystitis and cholangitis associated with the formation of membrane or casts of the bile passages. The disease simulates in most respects ordinary cholelithiasis, in which a gall-stone is passing through either the cystic or common bile duct, the pain and other evidences of such a condition being quite marked. Occasionally in these cases, when the stools are being searched for gall-stones, there have been found either distinct membranous casts shaped like the gall-bladder, or flakes of membranous material. The condition is usually associated with membranous enteritis, but there are, in addition, the symptoms of gall-stone colic and consequent jaundice, caused by the passage of solid material through the inflamed ducts.

The disease may be associated with gall-stones, or may follow after a long history of cholelithiasis.

As a rule the diagnosis is impossible unless membrane be found in the motions, and the case will be considered to be due to cholelithiasis. This mistake is of less consequence since the treatment for the two conditions is the same. If, under the exhibition of saline aperients and careful regulation of the diet, the symptoms do not abate, cholecystotomy should be done, any gall-stones present removed, and drainage of the cyst established. At the same time any adhesions of the biliary apparatus to the surrounding viscera should be broken down. After recovery from operation the patient should be advised so to regulate his life as to diminish the risk of the occurrence of any gastro-intestinal disturbance or of cholangitis.

CHOLANGITIS

Acute Catarrh of the larger Bile Ducts.—Catarrhal jaundice, so-called, is an affection which, for the most part, occurs in young persons, and usually results from dyspepsia or exposure to cold, but it may take origin from other causes such as pneumonia, the infectious fevers (especially typhoid), and cancer of the liver. That this condition is due to catarrh of the larger ducts is probable, though this has been disputed.

It is rare that uncomplicated cases succumb, but in some, where post-mortem examinations have been made, there has been found swelling of the mucous membrane, and plugs of mucus in the ducts have not infrequently been discovered. In addition, it is said, it can be seen that no bile has passed over the mucous membrane for some days, as all colour has disappeared from the affected part of the tube. Where death has taken place in cases of typhoid fever complicated with jaundice there has frequently been found unequivocal evidence of inflammation of the mucous membrane lining the bile passages. The catarrh is most marked in the common duct and gall-bladder, and gradually fades off in the hepatic ducts.

Judging from the appearances found post-mortem, as well as from the clinical histories, it may be inferred that the inflammation usually spreads from the duodenum. Even a very slight inflammation at the termination of the common duct in the posterior wall of the duodenum would suffice to block the passage of bile, since it is secreted under very low pressure.

Ordinarily catarrhal jaundice is preceded by some evidences of gastro-intestinal catarrh, either in the shape of a mild attack of dyspepsia with coated tongue and loss of appetite, or as an attack of nausea and vomiting with or without diarrhœa. In a day or two slight discoloration of the conjunctivæ appears, and the icterus increases for a week or ten days, thereafter gradually subsiding. Generally the patient feels out of sorts, but it is not usual for much constitutional disturbance to take place.

Where the catarrhal condition in the ducts complicates some other disease the course of events will depend on the nature of this illness.

In the majority of cases, where the patient is young and there is preceding gastro-intestinal disturbance, there will be little difficulty in arriving at a correct diagnosis in a case of simple catarrhal jaundice, but it should be remembered that slight jaundice is often a comparatively early symptom in cancer of the liver, being then usually due to associated cholangitis.

The possibility of the disease being acute yellow atrophy of the liver should be kept in view, and indeed during the first week of this disease there may be no signs pointing to the gravity of the condition, and its course may exactly simulate an attack of simple jaundice. In the later stages of the disease, however, the urgent gastric symptoms and the associated delirium, with the rapid pulse and subnormal temperature, taken along with the progressive diminution in the hepatic dulness and the appearance of leucin or tyrosin, or both, in the urine can leave no doubt as to the diagnosis.

In jaundice due to cholelithiasis the presence of pain and tenderness, and the previous history of gall-stone colic or "spasms," will readily lead to a definite opinion being given, but if, as sometimes happens, there be no marked pain the icterus will be found to pass off within a few days.

The jaundice of hypertrophic cirrhosis may closely simulate that from simple catarrh, but in this form of cirrhosis there is enlargement of the liver and usually other symptoms, such as ascites, pointing to the real origin of the icterus, while the more advanced age of the patient and the previous history of alcoholism will help to prevent a mistake in diagnosis.

Generally little treatment is necessary in order to get rid of simple catarrhal jaundice. Since the infection originates in the duodenum, and is kept up by the condition there, it will be well to give some mild purgative such as calomel or a saline. The food should be of such a character as to be readily digested and not subject to early fermentative changes; no alcohol should be given.

Large rectal injections of hot water at a temperature of from 70° to 90° F. have been recommended, and are supposed to act by inducing active contractions of the gall-bladder, which expel the mucus blocking the common duct.

Chronic Catarrh of the larger Bile Ducts.—Though simple acute catarrh may assume a chronic phase, this is unusual, and chronic cholangitis, giving rise to symptoms, is usually due to some other cause. Of these the most common is probably the presence of gall-stones, but in many cases of cancer of the liver the jaundice is, in the earlier stages, due in the main to the catarrhal condition of the mucous membrane of the bile ducts, which is invariably present at all stages. Similarly, when jaundice is present in cases of hydatid of the liver, or of hepatic abscess, it is more frequently due to an inflammatory swelling of the ducts than to pressure on them by the tumour.

Where the process is merely a continuation of an acute catarrh the symptoms will be slight, consisting mainly of more or less icterus and some gastro-intestinal disturbance. The persistence of the jaundice may lead to a suspicion of serious organic disease, especially cancer, but as a rule there is not much loss of flesh or strength, and the jaundice does not progressively deepen as it does in malignant disease. Moreover, the symptoms will, more or less readily, yield to appropriate treatment in the simple cases, whereas in malignant disease there will be at the best only temporary amelioration.

Where the catarrh has been caused by gall-stones and persists after these have been passed, there is apt to be present in the ducts thick ropy mucus which has difficulty in passing along the narrowed ducts and often gives rise to slight attacks of pain. Occasionally, as in cholecystitis, the mucus becomes much inspissated, and then when passing gives rise to pain resembling in almost all particulars that due to the passage of a gall-stone. So marked may be the resemblance, that in some cases it is not possible to differentiate between the two classes, but as a rule in cholelithiasis there is more marked tenderness, and for the most part some evidences of adhesions of neighbouring organs.

Chronic catarrh with or without jaundice should be treated, along the same lines as the acute affection, by salines (the best being the natural Carlsbad water), light diet, and regular exercise. With great care massage may be employed, and in the absence of gall-stones will probably be beneficial, but if there be any suspicion that the condition is directly due to cholelithiasis this treatment should be avoided. In the event of pain being present, topical remedies in the shape of hot fomentation, or the ingestion of a tumblerful of water as hot as it can be borne, will probably be sufficient; but it may be necessary occasionally to use sedatives. We have found half a drachm of spirit of ether in chloroform water, repeated every quarter of an hour if necessary, most effectual; but it may be necessary to give morphine hypodermically.

Unless cure or very marked relief follows this treatment within a couple of months, in all probability the best thing to do is to drain the gall-bladder and ducts by performing cholecystotomy, when if any gall-stones are present they may be removed.

Whether any calculi are found or not, the tube should be retained in the gall-bladder for some time; indeed, in the absence of stones it will probably be necessary to drain for a longer period. The best index to the time for withdrawal of the tube is sterility of the discharge; but roughly, it should be retained until the exudate is thin and contains little or no bile, as it is then clear that the normal secretion has been reached and the common duct is quite patent. After removal of the tube general treatment like that outlined above should be continued for some considerable time.

Infective Cholangitis.—In all forms of catarrh of the bile ducts there is probably some degree of microbic infection, and the peculiar symptoms which differentiate so-called "infective cholangitis" from ordinary catarrh are probably to be attributed to the occurrence of intermittent complete stoppage of the evacuation of the inflammatory products into the duodenum.

In the great majority of cases this will be found to be caused by gall-stones in the common duct either in the shape of a single stone "floating" in the duct and acting as a ball valve, or of multiple stones becoming impacted probably from some temporary swelling of the walls of the channels. Occasionally, however, malignant disease, either of the head of the pancreas and involving the orifice of the common duct or of the liver, is associated with infective cholangitis.

The symptom which specially characterises infective cholangitis is the occurrence of ague-like seizures, occurring at irregular intervals, and associated with remittent jaundice. The icterus in the interval between the attacks rarely completely disappears, though it may diminish so much as to be evident only on careful examination of the conjunctivæ in good light.

In a typical case there will be a more or less definite history of attacks of gall-stone colic, extending over, it may be, many years, and often without any marked degree of jaundice. Then comes a more severe attack, lasting longer, and followed by definite jaundice, probably denoting that the calculus has passed from the cystic into the common duct. After a short interval, during which the jaundice may have quite cleared, this is followed by another cholelithic seizure, accompanied by a rigor and all the symptoms of an attack of ague. Following on this there is jaundice which persists, though less in degree. This sequence of events recurs at irregular intervals, but in the majority of cases tends to return gradually more frequently, while the attacks become more severe. Even in the milder cases the strength of the patient progressively declines, and in the graver cases he may lose weight as quickly as if he were suffering from malignant disease of the stomach.

As a rule, this condition is not recovered from spontaneously, but occasionally the stone passes into the duodenum, and the patient gets well. It is, however, much more common for the course to be downward, the patient either succumbing to the repeated attacks of pain and poisoning, or to some complication of the disease, such as suppurative hepatitis, perforation of the ducts and peritonitis, endocarditis, or some pulmonary inflammation.

The diagnosis of this condition will not usually give much difficulty, at least in this country where ague is rare. The history of cholelithic colic extending over some time, the pain specially localised in the neighbourhood of the gall-bladder and ducts, and the tenderness most marked midway between the xiphoid and the umbilicus, together with the progressive deterioration of health and the remittent character of the icterus, all help one to an accurate opinion. It is not common to find the gall-bladder distended, as it has usually become thickened from previous chronic inflammation, nor is it the rule for the liver to be enlarged, though if the suppurative condition extend into the finer ducts there will soon be some degree of hepatic enlargement. From the beginning, however, tenderness in the right hypochondrium and epigastrium is present.

Treatment should not be put off when once the diagnosis is at all established, as in the great majority of cases palliative treatment is of no avail, and valuable time will be lost since the patient will progressively lose strength.

If possible the cause should be removed; but should this prove impossible, or the condition of the patient be such as to render it undesirable, the ducts should be drained and the cause removed by a further operation.

There can, however, be no doubt in the minds of those who have observed many cases that it is better to anticipate the complication, and as soon as medical measures, after a fair trial, have failed, to remove the gall-stones by surgical means.

Suppurative cholangitis, though in the great majority of cases caused by the presence of gall-stones, occurs also in association with cancer of the ducts, hydatid disease of the liver, and typhoid fever, and a number of cases have recently been reported in which the condition seems to have taken origin from the presence of ascarides in the common duct.

Associated with the causation in some obstruction in the ducts there comes dilatation to a greater or less degree of the lesser ducts in the liver, which also become very much inflamed. The whole liver enlarges rapidly, and may assume enormous proportions. Post-mortem the liver is large, and the ducts dilated and containing pus, while distributed through the liver there are usually found small abscesses of irregular shape.

Symptoms.—In the greater number of cases there will be a well-marked history of gall-stone colic, extending over a more or less lengthened period before the onset of acute symptoms, and it may be that there has been infective cholangitis; but where the disease is due to cancer, hydatid disease, typhoid, or any of the less common causes, the onset of the suppuration in the bile channels may be preceded by no local hepatic symptoms.

From whatever cause arising, the onset of suppuration is usually announced by a rise in temperature, with or without rigors or sweating, and this is followed by hectic temperatures, with rigors and profuse respiration.

Unless the attack has begun with a gall-stone colic, there may not be much pain at the beginning, but it is rare for some pain not to be present in the later stages. As with pain, so with tenderness; at first there may be little or none, but when the inflammation extends to the peritoneal covering of the liver the right hypochondrium becomes progressively more painful to the touch. Jaundice is present from the onset, and may become intense, but death as a rule takes place before this occurs. The liver enlarges uniformly, and may so increase in bulk that the lower margin descends below the umbilicus. The gall-bladder may enlarge, but as a rule it has become thickened and shrunken before the onset of the acute symptoms.

When the inflammatory disturbance implicates the peritoneum there come the usual symptoms of local peritonitis, viz. localised abdominal distension, with tenderness, and it may be vomiting. The pulse tends to increase in frequency and to lose in strength, the patient ultimately assuming the typhoid condition with dry tongue, shrunken features, rapid weak pulse, and great prostration.

In two cases of generalised suppuration in the liver (without any pulmonary complication) recently seen, the much greater proportional increase in the rate of respirations as compared with that of the pulse was a noteworthy feature, in each case the pulse-rate having gone up only 50 per cent, while the respirations had doubled in number.

Occasionally the disease pursues a subacute course, and then may end in a localised hepatic abscess, but this is extremely rare.

Death usually takes place from exhaustion, but may be accelerated by the occurrence of various complications, of which the commonest seem to be pyæmic abscesses, pleurisy, pneumonia, and infective endocarditis.

Pylephlebitis pursues a similar course, and it may not be possible to distinguish it from an attack of suppurative cholangitis unless there be a definite history pointing to the existence of some focus of suppuration in the parts drained by the portal vein.

In the earlier stages where the diagnosis is not clear the patient should be given 5 grs. of calomel, followed by a seidlitz powder, and local warm applications should be used, while if the pain is extreme it will be necessary to give sedatives; but whenever the case is made out clearly to be one of suppuration in connection with the bile passages, operation should be resorted to, since it is only in the early stages that one can reasonably hope for complete recovery.

As a rule it will be advisable only to attempt to give exit to the inflammatory products, and to establish drainage by performing cholecystotomy, leaving the cause to be removed at a later time. But if the patient be in fair condition, and the exciting cause of the malady can readily be reached, that should be got away at the same time.

If performed early, the relief to tension and the evacuation of the septic contents of the gall-bladder and bile ducts should give a very fair probability of complete cure, but even in the later stages, unless indeed the patient is moribund, it is well to seek relief by drainage, as some cases that appeared almost hopeless have recovered, and in any case the symptoms will be ameliorated.

Drainage should be kept up till the discharge becomes sterile.

Adhesions

Among the most distressing sequelæ of gall-stones, when these have been present for a long time, are the results which follow on the contraction of inflammatory adhesions to neighbouring viscera. The organ which is most frequently involved is the stomach, as the proximity of the duodenum and pylorus to the cystic duct renders it specially liable to suffer.

Usually the pylorus itself is tucked up closely to the cystic duct by adhesions, often so short as to render it a matter of extreme difficulty to separate the two without perforation of either viscus; but, not infrequently, there is merely some kinking of the pyloric extremity of the stomach or of the duodenum by adhesions to the gall-bladder or cystic duct.

Less commonly the whole, or part, of the lesser curvature of the stomach becomes adherent to the under surface of the liver.

Whichever of these conditions is present, there follows the whole series of symptoms which arise when, from any cause, the stomach is unable to empty its contents into the duodenum within the normal time. When the stomach wall over a considerable area is adherent to the liver, but there is little or no implication of the pylorus, the symptoms will only amount to some degree of dyspepsia and a feeling of discomfort in the epigastrium, coming on shortly after food and lasting for several hours, since there is no actual obstruction to the outflow from the stomach, but only interference with the peristaltic wave. When, however, the pylorus becomes constricted by surrounding adhesions, or the exit from the stomach is obstructed by kinking, there follows dilatation of the stomach, giving rise to the well-known symptoms of that disorder which will prove most intractable to ordinary medical remedies, and even to lavage. If left untreated by operation this condition exhibits no tendency to improve; but, as a rule, despite the most careful and continued treatment, the condition of the patient becomes progressively worse, the stomach dilating so as to reach almost to the pubes, vomiting, even after liquid food, taking place after each meal, and the patient becoming worn out from the combined effect of defective nutrition and continued pain. The frequency of this condition is not yet quite realised by most practitioners, though those who see much of gall-stone work must have noted the large proportion of cases in which it occurs to such an extent as to be readily diagnosed, before operation, by the usual physical signs of dilatation of the stomach.

It should be noted that, when the pylorus is adherent to the cystic duct, and there are biliary calculi in the duct or gall-bladder, the pain of a cholelithic seizure may radiate to the *left* subscapular region instead of to the right.

Less commonly, but still not infrequently, the colon is obstructed to a greater or less degree by the presence of short adhesions to the gall-bladder, or by larger bands resulting from localised peritonitis so pressing on the bowel as to interfere with the circulation of its contents. As might be expected, the middle part of the transverse colon is the usual seat of obstruction, but we have seen the hepatic flexure blocked, and in one case recently treated for acute obstruction there were two marked bands, one situated almost at the hepatic flexure, the other within a couple of inches of the cæcum.

Where the colon is involved the symptoms are as a rule less severe than when the stomach is affected, but, when bands have formed, acute intestinal obstruction may rapidly develop. As a rule, however, there are only evidences of chronic intestinal obstruction, showing itself by attacks of paroxysmal colicky pains in the abdomen, occurring at irregular intervals and associated with constipation, or constipation alternating with diarrhœa. Vomiting is not commonly present, but, when the kinking is acute or the bowel is constricted by bands, this may be a prominent feature. In many of these cases during the attacks of pain careful examination will reveal the cæcum contracting in its efforts to force the contents of the colon through the stricture or past the kink; and at times large fæcal concretions can be felt on the proximal side of the obstruction.

In all these respects the case may closely simulate cancer of the large intestine, but in the latter there is more rapid deterioration of the general health, and since the stricture in malignant disease is most often in the sigmoid flexure or rectum, the motions tend to be flattened, and there is straining and the presence of mucus and blood in the stools.

When gall-stones give rise to local peritonitis so marked as to lead to obstructive symptoms, there are always long-continued and repeated attacks of colic, so that the past history is of great importance in arriving at a correct conclusion in any individual case, though it should be remembered that cholelithiasis and cancer may of course be present at the same time.

In most cases of adhesions there is more localised tenderness than when the obstruction is due to new growth, but the attacks of paroxysmal pain may be as severe in one case as in the other.

Where adhesions are diagnosed, or where, after a history of repeated attacks of gall-stone colic, symptoms arise which might be referable to adhesions, and which do not subside under medical treatment sufficiently long continued, operation should be undertaken, and the adherent viscera separated. As a rule there will be gall-stones present, but, in any case, it will probably be advisable to perform cholecystotomy and drain the gall-bladder to get rid of the catarrhal condition of the mucous membrane, which is almost invariably present. In order to prevent the recontraction of the adhesions the omentum should be tucked up between the gall-bladder and the contiguous viscera.

After recovery from operation the patient should be advised to continue medical remedies directed to prevent catarrh of the duodenum and bile duct, as has been advised after cholecystotomy for gall-stones.

ULCERATION AND ITS SEQUELÆ

Ulceration of the gall-bladder and ducts is probably fairly frequent, but if uncomplicated does not usually give rise to symptoms of much importance. Although cholelithiasis is the most frequent, typhoid fever and cancer are quite common causes, and tubercular disease and cholera also produce ulceration.

As has already been pointed out, pyrexia when present in empyema of the gall-bladder is probably always associated with ulceration, and due to absorption of septic products through the ulcerated surface.

The importance of ulceration, however, depends more on its sequelæ, the most important of which are adhesions (already considered), perforation, fistula, peritonitis, hæmorrhage, and stricture.

As the ulceration extends through the wall of the cyst or its ducts, there is set up local peritonitis, which usually induces adhesions to the neighbouring viscera, and thus shuts off the general peritoneal cavity, but occasionally no adhesions are formed, and perforation takes place. On account of the fact that the bile in cases of cholelithiasis is invariably infected, this event is always very serious, since general septic peritonitis is almost certain to ensue, and unless quickly treated will end fatally. Rarely the perforation is followed by a localised peritonitis, which shuts off the general peritoneal cavity, and allows of the formation of an abscess which runs a course similar to that of an abscess due to appendicitis.

The symptoms of perforation of the bile passages are those of perforative peritonitis from any cause, with, as a rule, a history pointing to the presence of gall-stones. A sudden pain beneath the right costal margin followed by collapse and succeeded by vomiting, general distension, and a rapid pulse, form the chief features of the disease. If the extravasation is extensive there will be signs of free fluid in the peritoneal cavity, and jaundice, if not present before, usually appears within twenty-four or forty-eight hours from absorption of biliary pigment by the peritoneum. Death takes place usually within a few days, but cases have been reported where life was prolonged into the second or third week.

As a rule the condition of the patient does not warrant more than that the abdomen should be opened, the extravasated material removed by sponges, and satisfactory drainage established; but, if his condition be sufficiently good, the perforation should be found and stitched, gall-stones if present removed, and the gall-bladder drained separately from the general cavity of the peritoneum.

FISTULA.—Fistula is a not infrequent complication of ulceration of the gall-bladder and bile ducts. Much more frequently the communication is between the surface of the body or the cavity of one of the hollow viscera and the gall-bladder, but communication with the larger ducts is not uncommon. In the direct variety, where the channel is formed directly through local adhesions set up by the advancing ulceration, the communication is most frequently with one of the hollow viscera; whereas, in the indirect variety, caused by the formation of an abscess outside the gall-bladder and a channel forming from this, the fistula more usually opens on the surface of the body.

As might be expected from the anatomy of the parts the structures most commonly affected are the duodenum and colon; but fistulæ have been described between the biliary passages and the stomach, the jejunum, the ileum, and the pelvis of the right kidney, while a number of cases have been described in which gall-stones have perforated into the thorax.

Biliary cutaneous fistula, like the other forms of fistula in connection with the bile passages, is most commonly a sequel of gall-stones, but may result from any of the causes which give rise to perforation of the bile channels, especially when the perforation leads first to a localised abscess. When arising from suppuration in connection with the gall-bladder or bile ducts the fistula usually opens near the umbilicus, the pus following the course of the obliterated umbilical vein, but the discharge may occur at any part of the abdominal wall. Occasionally a fistula persists after operation where the obstruction in the ducts has not been overcome, or the gall-bladder has been stitched to the skin instead of to the peritoneum and aponeurosis. Their importance varies according as only mucus or bile and mucus are discharged, since in the former case only about one ounce of fluid is discharged daily, and this does not give rise to much inconvenience.

Mucous fistula occurs when the cystic duct is obstructed by the presence of a foreign body, or is occluded by stricture resulting from old ulceration.

So long as there is free exit to the secretion there will only be the inconvenience arising from the necessity of constantly wearing some dressing to absorb the discharge; but if the orifice of the sinus be allowed to close, the accumulation produces pain, and it is necessary under these circumstances for the patient either to wear a small drainage-tube and an absorbent dressing or to submit to operation.

Should operation be decided on, the course pursued will depend on the cause of the condition. If there be an impacted calculus in the cystic duct, this should be removed; but if the continuation of the discharge depends on stricture of the duct, the gall-bladder should either be excised or connected with the duodenum by means of a Murphy's button. As a rule, in the latter case, cholecystectomy will be safer, since the gall-bladder, under these circumstances, is invariably small, and cholecystenterostomy in a case where there is a shrunken gall-bladder is always difficult of performance.

Biliary fistula is a much more serious matter, because, in addition to the disability caused by thirty ounces or more of bile being discharged on to the skin daily, there is apt to be interference with the general nutrition.

This form of fistula arises from some obstruction to the flow of bile in the common duct, most commonly from an impacted stone, but occasionally from malignant disease of the head of the pancreas or of the common duct.

Where possible the obstruction should be removed; but when it arises from malignant disease this is usually impossible, and when due to gall-stone sometimes inadvisable owing to the weak condition of the patient. In the event of it being decided not to remove the obstruction, the best course open is to connect the gall-bladder with the duodenum, by performing cholecystenterostomy.

Before opening the peritoneal cavity in any case of biliary cutaneous fistula it is well to purify the sinus as well as possible by curetting the granulating track.

In a case definitely known to be due to a gall-stone in the ducts, before resorting to operative interference it may be well first to try for some time the effect of injecting olive oil, or a ·5 per cent solution of sapo animalis, by means of a flexible catheter introduced into the sinus, on to the obstruction two or three times daily, in the hope that by this means the concretion will be dissolved.

Biliary-Intestinal Fistula.—Fistula between the bile ducts and some part of the gastro-intestinal tract is a fairly common complication of ulceration due to gall-stones, and frequently arises without any overt manifestations, the ulceration proceeding slowly through adhesions into the lumen of the stomach or gut, the gall-stones being discharged and the fistulous track healing spontaneously. In several cases which we have seen, and in others reported, the first indication of anything having happened has been the onset of acute intestinal obstruction from the gall-stone becoming impacted low down in the bowel. But the process is not always accomplished without symptoms, such as pain over the liver, more or less jaundice, a fever of irregular character, with, it may be, some hæmorrhage into the stomach or bowel, while there is always the possibility of perforative peritonitis. Apart from the complications spoken of and the subsequent adhesions, the formation of such a fistula does not usually cause much inconvenience or give rise to any need for operative interference, but when the communication is with the stomach the passage of bile into that organ may lead to the necessity for active treatment. Rare forms—biliary-urinary, biliary-vaginal, biliary-thoracic, biliary-pulmonary, biliary-pericardial, biliary-mediastinal, biliary-pleural, biliary-retroperitoneal, and biliary-portal—have been described, but they are of extremely infrequent occurrence.

Stricture of the bile ducts, apart from that due to cancer, is, judging from reported cases, a much rarer condition than might be supposed. Though there is no reason why it should not be a sequel of ulceration arising from other causes than cholelithiasis, we are not aware of any case having been reported where it was not preceded by the presence of biliary calculi. It should be noted, however, that it may not declare itself until some time after the exciting cause has been removed, either by operation or through ulceration leading to the formation of a fistula through which the stones escape.

If in the cystic duct the only symptom present may be a gradually enlarging tumour in the gall-bladder region, with or without pain or uneasiness; if in the common duct, jaundice supervenes, at first being only slight, but ultimately becoming intense, the liver progressively enlarges, and if it be not shrunken from the continued irritation of gall-stones, the gall-bladder distends.

So far as we know, only one case of stricture of the hepatic duct has been described, and in it the symptoms were like those of stricture of the common duct, but with no distension of the gall-bladder. The case ultimately proved fatal from peritonitis due to ulceration of a stone into the peritoneal cavity.

Cases of stricture of the ducts can scarcely be diagnosed, but will mostly be suspected to be cases of impacted calculus, the condition only being recognised when the abdomen is opened.

In stricture of the cystic duct probably the best treatment is to remove the gall-bladder, but cholecystenterostomy may be performed.

When the obstruction is in the common duct, cholecystenterostomy or choledochenterostomy should be done if at all possible; if not, cholecystotomy will relieve the symptoms.

Unless there is great dilatation of the hepatic duct no operation is likely to be of much avail in stricture of that channel, but if it is sufficiently distended it might be feasible to connect it either with the gall-bladder or with the intestine by means of a Murphy's button. If at all possible the former measure would be the better, as the latter would probably be followed by suppurative cholangitis.

STRICTURE OF THE GALL-BLADDER, giving rise to an hour-glass shaped organ, occasionally occurs from ulceration of the gall-bladder, and may even proceed so far that the upper cavity is quite shut off from the lower. In such a case the upper sac should be amputated and the lower drained, as in ordinary cholecystotomy, after any concretions which may be present have been removed.

PERITONITIS of an acute kind may occur along with the ulceration apart from any gross communication between the interior of the bile passages and the peritoneum, the removal of the epithelial lining apparently permitting microbes to pass through the wall of the sac, but this is not common. Another uncommon complication of ulceration is *hæmorrhage*. Usually as the ulceration proceeds thrombosis takes place in the vessels, but occasionally severe hæmorrhage results, and several cases have been recorded in which the fatal issue seems to have been determined by loss of blood arising in this way.

TUMOURS OF THE GALL-BLADDER

Under this general head it is convenient to consider two very different conditions, viz. enlargement from distension and new growths affecting the gall-bladder.

The gall-bladder may, from a variety of causes, be so distended, apart from any new growth, as to be felt on palpation of the abdomen. The commonest cause is undoubtedly obstruction to the cystic duct by the impaction of a gall-stone or from hydatid disease, but it may follow on the blocking of the common duct by a stone or from pressure on the ducts exercised from without. The proportion of cases in which cholelithiasis is associated with distended gall-bladder is, however, comparatively small, as mostly, where gall-stones have been present for some time, they cause so much inflammatory thickening of the wall of the cyst and destruction of its mucus-secreting glands that it is incapable of much distension even should the ducts become blocked,—indeed it is much more common to find it shrunken and smaller than normal.

Where malignant disease is so situated as to occlude the larger bile passages, it is very much more common to find the gall-bladder so distended as to be palpable, because there has, as a rule, at least when the cause of the obstruction is situated in the common duct as it most frequently is, been no preceding interference with the glands studding the mucous membrane of the gall-bladder, and these continue to pour out mucus while the normal walls of the cyst readily yield to the increased internal tension.

A distended gall-bladder rarely reaches much below the umbilicus, but some have been found to fill the abdomen to such an extent as to have been operated on under the assumption that they were ovarian tumours, and in one case the fundus of the gall-bladder was discovered in a femoral hernial sac. Except in those cases where the obstruction is in the cystic duct, the contents are probably in all cases at first bile with a small percentage of mucus, but later the bile becomes absorbed, and only mucus is present unless acute inflammation supervenes, when muco-pus will be formed.

A distended gall-bladder is to be made out as a pear-shaped body passing downward and forward from the ninth costal cartilage towards the middle line just below the umbilicus and moving with respiration. Where there has been little local peritonitis it will be felt quite distinctly as a smooth, rounded, and tense tumour, the lower extremity freely movable from side to side, but becoming more fixed and less defined as the liver is reached. In such a case in a thin patient the mass can usually also be seen to move up and down with expiration and inspiration, but if there has been local inflammation this will not be so, and the tumour will be found much less defined and probably more fixed. If the gall-bladder is inflamed there will also be a greater or less degree of tenderness, whereas in simple enlargement the mass will be painless on manipulation.

Partly on account of its tenseness, but mainly by reason of its mobility, fluctuation cannot usually be obtained, while absence of dulness on percussion is not uncommon from the presence of coils of intestine overlying the tumour.

In some cases we have found it easier to be sure of the presence of a distended gall-bladder by making the patient assume the genu-pectoral position, when it can be readily felt lying on the flat hand placed on the right side of the abdomen.

The detection of variations in size from time to time is of great importance, as it practically invariably denotes that the cause of the obstruction is simple, whereas in malignant disease once distension has occurred it persists.

Jaundice may or may not be present along with the palpable gall-bladder, according to the site of the obstruction, but when present it is a grave sign by reason of the consequent interference with nutrition, and from the fact that the two conditions are more usually associated with malignant disease than with cholelithiasis.

Pain is not commonly marked after the onset; but the beginning of the condition, when due to biliary calculi, is usually ushered in by an attack of gall-stone colic, while when arising from malignant disease there is for the most part no severe pain.

The commonest error in diagnosis is to assume that a distended gall-bladder is a floating kidney, or a renal or supra-renal tumour, and the mistake is more readily made than might be expected. With care, however, this can usually be easily enough avoided. Where the obstruction is in the common duct there should be no difficulty, since the presence of jaundice will readily localise the site of the disease. When the obstruction is in the cystic duct the case is not so easy, but the fact that the tumour is continuous above with the liver, that its lower extremity only is movable, and its range of movement small, that it is pear-shaped and not kidney-shaped, that though movable from side to side it cannot be displaced downwards at all other than by the patient taking a deep inspiration, and that when left alone it does not tend to fall towards the lumbar region, should render the diagnosis clear. Should there have been attacks of severe pain at any time the different characters of renal and gall-bladder pain, both as regards site of origin and distribution, will help to define the case.

In case of difficulty, which will occur mostly in very stout persons, or in those who are unable to relax their abdominal muscles, examination with the patient under an anæsthetic may be necessary. Only rarely will it be necessary to apply Ziemssen's test, which consists in distending the colon with gas, and so displacing a kidney tumour into the loin, or a distended gall-bladder toward the liver at the junction of the right hypochondrium with the epigastrium.

Where the kidney or supra-renal tumour has become adherent to the colon this test if relied on may give a false impression, as the mass may be raised very much into the position usually assumed by a distended gall-bladder.

Primary malignant disease of the liver in its early stages may closely simulate distension of the gall-bladder in its physical characters, but the greater irregularity of the surface, the history of the illness, the rapid loss of weight and strength before the formation of a perceptible tumour, and the subsequent onset of jaundice, will assist an accurate diagnosis. Hydatid disease is more likely to give rise to difficulty, but it is not so well defined, is not pear-shaped, and is as a rule painless throughout its history, while generally fluctuation or the characteristic thrill can be detected.

Tumours of the intestine and of the pylorus should be kept in mind when examining a case of suspected gall-bladder tumour, but the associated symptoms will usually enable one to make the diagnosis. In this connection it should be remembered, however, that dilatation of the stomach is very frequently a result of the pylorus becoming adherent to the cystic duct in cholelithiasis.

The possibility of the tumour being a Riedel's lobe should be remembered, but when this abnormal projection is present it is usually farther to the right, its shape differs from that of a gall-bladder, being broadest at its upper extremity and tapering off downwards, while it is harder and firmer. At times, too, the gall-bladder can be felt to its inner side.

The treatment of distended gall-bladder will depend on the cause, but as a rule, when due to non-malignant disease it will be necessary to perform cholecystotomy, and at the same time remove the obstruction whether that be situated in the cystic or common duct. When due to stricture of the cystic duct the gall-bladder should either be removed, or connected to the duodenum by the operation of cholecystenterostomy.

New growth affecting the bile passages is almost always of the nature of columnar-celled epithelioma, but simple tumours have been found from time to time, and some believe that adenomata precede all primary carcinomas affecting the gall-bladder or ducts.

Primary cancer of the gall-bladder is not very common; but it does supervene on chronic cholelithiasis more often than is usually believed, as when the disease occurs it is quite commonly assumed to be primary malignant disease of the liver. More frequently the gall-bladder becomes affected with cancer by extension from neighbouring organs, and then the tumour differs in its histological characters according to the site of origin. When the gall-bladder alone is affected as a rule the wall is uniformly infiltrated before the disease is sufficiently marked to give rise to symptoms. Dissemination, otherwise than by local extension, is rare, but the glands in the lesser omentum usually are involved in the later stages. Mostly the liver is the organ first invaded, but where cholelithic symptoms have been marked it is not unusual for the pylorus, duodenum, or colon to be involved early on account of their having become previously adherent to the gall-bladder.

The diagnosis of cancer of the gall-bladder is always difficult in the early stages, and often impossible before exploration of the abdomen.

Almost invariably there is a history of attacks of gall-stone colic extending over a lengthened period, then comes progressive deterioration of health without much local manifestation other than a sense of discomfort in the gall-bladder region scarcely amounting to pain. As the disease progresses the pain becomes more marked and more diffuse, often extending toward the right scapular region.

In the early stages no tumour can be made out on palpation, but with the growth of the local disease a hard rounded mass comes to be readily felt below the costal margin, at first freely movable during respiration, but later becoming more fixed and nodular. Unless gall-stones are present there is throughout an absence of anything like marked tenderness.

Early in the disease there is no jaundice, but later jaundice is frequent, and may be due to extension of the disease along the cystic duct, to catarrh of the ducts, to invasion of the liver, or to pressure on the common duct by the enlarged lymphatic glands.

Associated with these symptoms may be others arising from the extension of the growth to the surrounding organs.

As the disease progresses, the general condition of the patient rapidly deteriorates until he succumbs to the malady.

Malignant disease may be closely simulated by inflammatory adhesions in the neighbourhood of the gall-bladder, but in the latter case there is not the same deterioration of health unless suppuration supervenes, when the elevation of temperature and marked local tenderness will afford a clue to the state of affairs.

Where the disease is limited to the gall-bladder, treatment by cholecystectomy affords a very fair hope of cure; and even if the liver be involved, in certain cases it will be found possible to remove the disease with a possibility of cure, and the certainty of amelioration of symptoms. In the majority of cases, at the present time, however, when the patient comes to the surgeon nothing but the alleviation of symptoms by the exhibition of sedatives can be hoped for.

Tumours of the Bile Ducts

When the gall-bladder is much shrunken from old-standing inflammation it occasionally happens that the common duct distends so markedly, as a result of obstruction low down, that it can be made out as a distinct tumour.

As a rule these cases have been diagnosed either as distended gall-bladders or as hydatid disease of the liver, and the true state of affairs has only been made out when the abdomen was opened. If such a case be due to an impacted gall-stone this should be removed, and the opening in the duct though which it is extracted stitched up, since the cases which have been treated by drainage of the duct have nearly always up to the present done badly. Where it is not possible to remove the cause, choledoch-enterostomy should be performed rather than choledochostomy, since a permanent biliary fistula will follow the latter procedure, while the chance of recovery from operation, judging from published results, is distinctly less than when the bile stream is short circuited.

Simple *new growths* of the bile ducts occur, but are very rare; malignant disease, though not at all a frequent condition, is much more common.

Any part of the larger ducts may be involved, but the tumour is most frequently situated in the lower part of the common duct.

Practically in all cases cancer of the ducts is preceded by cholelithiasis, and this renders the diagnosis difficult, as the symptoms of both conditions are usually present.

The growth is usually annular, and histologically is a columnar-celled carcinoma. The special symptoms depend on the fact that the outflow of bile is readily obstructed; and thus when situated in the common duct malignant disease gives rise early to jaundice which progressively deepens, the liver gradually increases in size, and the gall-bladder distends, unless previously shrunken as the result of the presence of gall-stones.

Infective or even suppurative cholangitis not infrequently supervenes, and gives rise to the symptoms already described. Pain may be present, even though the gall-stones have passed; but, where none are present, there is as a rule little or no tenderness, but only a feeling of discomfort on deep pressure in the epigastrium.

Should the cystic duct be the site of origin of the disease the gall-bladder will distend, but jaundice will not be present to any degree until the growth has by extension invaded the common or hepatic duct, though even in the early stages slight icterus may be noted from a catarrhal condition extending from the stricture. Almost invariably gall-stones are present in the gall-bladder or cystic duct, and these may give rise to pain of a paroxysmal character and to tenderness.

The hepatic duct is comparatively seldom the first to suffer, but when it does the symptoms will be those due to obstruction of the common duct, but for the fact that of course there will be no distension of the gall-bladder.

It should be remembered that obstructive jaundice frequently arises from the extension of malignant disease from neighbouring organs, especially from the head of the pancreas, and while it may be impossible to differentiate the two conditions, as a rule there will be some symptoms pointing to the origin of the disease outside the ducts.

Only in tumour of the cystic duct at or shortly after the onset of the mischief can radical treatment by removal of the disease be attempted.

Where the hepatic duct is involved, surgical interference is practically of no avail; but when the obstruction is in the common duct, two courses of surgical treatment may be followed with relief to symptoms. Either cholecystotomy may be performed and a permanent biliary fistula established, or cholecystenterostomy may be done. Should the patient be in fair condition, and the gall-bladder distended or normal in size, the latter alternative should be adopted; but if he is weak, this operation becomes very dangerous, since it is necessarily more prolonged than a simple cholecystotomy, and in the event of the gall-bladder being less than its normal size should not be attempted.

Too often, however, before the patient comes under the surgeon's care he is so reduced that no operative measures are justifiable, and only remedies directed to the amelioration of his sufferings can be advised.

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Galvanic Cautery.

See also Cautery.

The Galvanic Cautery is an instrument for burning tissues, the heat being produced by a current of electricity.

Principle of Construction.—A piece of metal of relatively high resistance—platinum being most commonly used—is placed in a circuit of ordinary copper wire. The copper wires are carefully insulated and bound together, being prolonged into the handle of the instrument. When the current is allowed to pass, the platinum, offering resistance, becomes heated, and thus forms the cautery. The handle is provided with a key by which the current may be easily closed and opened.

Source of Current.—The current may be obtained from (1) a battery; (2) an accumulator; or (3) a lighting main.

1. *Battery.*—If a battery be employed for this purpose it must be different from that used for galvanisation or for electrolysis. In the latter cases high electromotive force, that is, high voltage, is required to overcome the high resistance of the human body, whereas a strong current cannot be borne. On the other hand, a strong current is required to render incandescent a piece of platinum of the thickness necessary for a cautery, and to produce such a current and maintain it constant, even for a short time, large cells are necessary; but as the resistance of platinum wire is very small relatively to that of the human body, a small electromotive force is required, therefore few cells.

A battery to be used for the sole purpose of providing current for a galvanic cautery should consist of from two to six bichromate cells of large size, the square shape being preferable to the bottle shape because it admits of the plates being further apart, which secures greater constancy of the current. These cells should be connected "parallel," *i.e.* zinc to zinc and carbon to carbon, for by this arrangement the internal resistance is diminished and a stronger current is yielded. For the ordinary small cautery the battery must be able to produce a current of at least eight ampères.

2. *Accumulator.*—Generally the most convenient and the most reliable and constant source of current is the accumulator. It does not easily get out of order, and may be simply adapted for use either with the cautery or for a small electric lamp. Two or three secondary cells, each giving two volts, enclosed in a strong oak case, will be found most convenient for general purposes.

3. *Lighting Main.*—Both continuous and alternating sources of electrical supply may be employed for cautery work.

With a *continuous current* a suitable resistance, consisting of spirals of thick wire, must be provided. As the pressure in these mains varies from 100 to 250 volts a large amount of energy is lost, seeing that only two to four volts are required in the cautery. With a properly constructed resistance all dangers from accident are avoided, and, as the current is usually required for a very short time, the cost is not serious.

An *alternating current* affords a very simple and economical means of heating a cautery. All that is necessary is to insert in the circuit the primary of a small transformer, the secondary of which is wound so as to produce a large current at a small pressure. The strength of the current in the cautery is regulated by the position of the two coils relatively to each other. An excellent transformer, suitable for this purpose, is that devised by Woakes.

FORMS OF INSTRUMENT IN USE.—In work on the nose and throat and in dental work are found the commonest applications of the galvanic cautery. The handle devised by Dr. Schech is the one most commonly used. To this any of the platinum points, or "burners," of which there are many forms—probe-pointed, flat, spear-shaped, etc.—differing in size and shape, may be fitted; or a wire, arranged as a snare or écraseur. For the latter purpose the ordinary steel wire, of various thicknesses, which is used for pianos, is quite suitable.

When a larger heated surface is required a piece of porcelain may be employed, introduced within a loop or spiral of platinum wire, which with the wire becomes heated when the circuit is closed.

DISEASE IN WHICH THE GALVANIC CAUTERY IS EMPLOYED.—*Throat.*—In hypertrophic pharyngitis, for the destruction of hypertrophied tissue. It may sometimes be applied to hypertrophied tonsils. In the larynx it has been employed for the destruction of small neoplasms; or, as a snare, for the removal of larger pediculated tumours.

Nose.—Again, for the destruction of hypertrophied tissue causing obstruction of the nares. Here the flat burner is employed; but the snare may also be used to remove large masses, especially when they are attached by broad bases, because the hot wire makes for itself a furrow, and obtains thus a hold which the cold wire may fail to do. It is also useful for cauterising pedicles of polypi removed by the cold snare or by forceps. And in cases of epistaxis due to an unhealthy condition of the mucosa its application, at a dull red heat, is very valuable.

Ear.—Here the applications are fewer, but the galvanic cautery may be used for the removal of redundant tissue in the meatus, or for perforating the drum membrane, in rare cases. It has also been used for destroying foreign bodies impacted in the meatus, as peas, etc.

Gynecology.—Most frequently used as a snare for amputation of a hypertrophied cervix uteri. But it is also sometimes employed as a cautery for destroying urethral caruncles.

Genito-urinary Surgery.—For the radical cure of the symptoms which arise in cases of enlarged prostate a special apparatus has been devised by Professor Bottini. It requires a very strong electric current.

General Methods, etc.—Local anæsthesia should always be induced before the cautery is used, by means of pledgets of cotton wool, saturated in cocaine (20 per cent), and placed in contact with the affected part for a few minutes before the application is made.

The operator must be careful to test the apparatus before using it. All the screws and connections must be clean and firm, and the wires used for connections strong and well insulated. The current must be gradually strengthened till the burner is brought to a white heat. (In contact with the tissues this will just procure the necessary degree of redness.) If this be not done the point may fuse whilst in use, causing inconvenience and delay. It should be borne in mind when the snare is used that the wire requires less current to heat it than do the platinum points, and that the thinner the wire the less the current required. If any readjustment be necessary the current from the battery should be cut off before the wires are touched by the fingers.

When the instrument has thus been tested the terminal is to be applied cold to the desired point, and the current closed by means of the key on the handle. It must be removed from contact with the tissue while still hot, or it will stick, and produce tearing and bleeding in removal. The connections of the points are easily melted if the current be kept closed too long. After removal from the patient the point should be cleaned by heating it to burn off adhering tissue.

With regard to *after-treatment*, it is important to allow sloughs to fall off before any further application of the cautery is made. An interval of a week or two should therefore elapse between the operations in cases where several applications require to be made. During this time the patient must be exposed as little as possible to septic influences, and this may be aided by the use of antiseptic ointments, sprays, or lotions applied to the affected part.

Ganglion.

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Definition.—The word ganglion denotes a localised swelling in connection with a tendon sheath. It is derived from γάγγλιον, a hard swelling. Such a swelling is primarily cystic in nature, though it may happen that the cavity of the cyst becomes so encroached upon by the fibrous tissue of the wall as to render the tumour almost completely solid.

Causation.—There is still a considerable diversity of opinion as to the exact etiology of cysts in relation to, or in connection with tendon sheaths. They may arise from—(1) The hernial protrusion of the synovial membrane of the sheath through the outer fibrous tunic, (2) a colloid degeneration of the cells of the synovial fringes, (3) the dilatation of the sub-synovial crypts or follicles of the tendon sheath or adjacent joint cavities, (4) a chronic teno-synovitis, and possibly (5) tuberculous affection of the synovial membrane of the tendon sheath.

There are other cystic swellings that may occur in the same regions as those that are the common sites of ganglia, which, although closely resembling true ganglia, have their origin quite apart from a tendon sheath. Among these may be mentioned bursæ, hygromata, and hydatid cysts.

True ganglia are present more frequently in the female than in the male sex, and are most prevalent from ten to thirty years of age.

Certain occupations and pursuits are apt to be predisposing causes. Musicians, laundry workers, and others who exercise greatly the muscles of the fingers, are peculiarly likely to be affected.

SITUATION.— Ganglia are most frequently found associated with the tendons of the extensor and flexor muscles of the wrist, fingers, ankle, and toes, and are particularly liable to affect the tendons of the extensor communis digitorum and the extensor indicis, though the extensors of the thumb are almost equally involved. All of these tendons, it will be observed, are on the radial side of the wrist. On the dorsum of the foot the tibialis anticus and the extensor proprius hallucis are the tendons that are most commonly the source of origin. Occasionally ganglia may arise about the insertion of the hamstrings, and still more rarely about the elbow.

PATHOLOGICAL ANATOMY.—The tumour is covered, at any rate primarily, with healthy skin, to which it is not adherent. As a rule there is a fibrous cyst wall varying in its thickness. This is more or less perfectly lined by endothelial cells, capable of secreting a characteristic fluid. The colloid material found within the sac possesses the consistence of honey that has been strained, and is usually colourless and alkaline in reaction. It is distinctly proper to ganglia, not being found in any other cavity, being as characteristic of them as the contents of a sebaceous or of a dermoid cyst are for such. It is altogether different from bursal or ordinary synovial fluid.

Ganglionic cysts are attached in nearly every case, as can be demonstrated by careful dissection, to one or more of the neighbouring tendon sheaths, and in addition to the capsule of the joint which is adjacent to them. Intercommunication, however, between the synovial cavity of the tendon sheath or of the joint and the interior of the ganglion is the exception rather than the rule.

SIGNS AND SYMPTOMS.—*Signs.*—Ganglia occur as prominent swellings, varying much in size, but seldom attaining a greater magnitude than that of a walnut. Sometimes they are distinctly globular or hemispherical in shape, and at others flattened. To the touch they are generally densely hard, hence the origin of the term "ganglion," but they may be of a softer character, and they may fluctuate readily, while, when tense, and owing to the nature of the contained fluid, fluctuation cannot always be obtained, yet they are in every instance truly elastic. When present on the palmar aspect of the fingers in connection with their flexor tendons, they may be so firm as to be mistaken for solid tumours. They may become apparently diminished in size according to the position assumed by the joint near which they lie. When the wrist is their situation they will be more tense and prominent when this joint is flexed ventrally, and seemingly almost disappear when it is again fully extended. With the motions of the fingers or toes there may be some movements communicated to the ganglionic swellings.

Symptoms. — The possessor of a ganglion usually complains of the deformity caused by the swelling, and attributes pain to it especially on putting into action the muscles upon the tendons of which it appears to be. This pain may be so severe as to prevent the sufferer from firmly grasping an object in the hand, or of walking in boots with comfort. A sense of weakness of the part is almost always present. Occasionally the actual pain is referred to some distance up the limb. In hysterical or neurotic patients extreme discomfort will result from the condition.

DIAGNOSIS.—(*a*) From other cystic swellings: 1. Bursæ; 2. Aneurysms; 3. Synovial cysts.

(*b*) From solid swellings: 1. Fibromata; 2. Lipomata; 3. Tuberculous growths; 4. Gummata; 5. Osteomata; 6. Chondromata; 7. Sarcomata.

In the diagnosis from bursæ some difficulty may be experienced, for both varieties of swellings may exist in similar regions, and both may be associated with tendons. Bursæ, however, are as a rule over bony points, while ganglia have no connection with such. Bursæ contain typical bursal synovial fluid, while ganglia are filled with their characteristic colloid material. It is usually in the case of a ganglion in relation with the flexor carpi radialis tendon sheath that a possible error in diagnosis can occur. Here there may be communicated pulsation to the swelling, and if there has been a history of injury, a somewhat close resemblance to a traumatic aneurysm presents itself. Moreover such aneurysms of the radial artery are not by any means rare. The chief point of distinction lies in the fact that if the swelling is displaced a short way from the line of the vessel the pulsation ceases, and that an exploring needle, introduced only in such cases as will be submitted to operation, reveals the contents to be in one case blood and in the other the usual colloid material.

Synovial cysts may be almost impossible of diagnosis unless their contents are seen, when the synovial fluid will not have the characters of the ganglionic. In the majority of instances the fact that fluctuation can be obtained in a ganglion serves to distinguish it from any of the solid swellings enumerated above.

TREATMENT.—This may be either non-operative or operative.

Non-operative Treatment.—1. Application of the Tincture of Iodine.—For a considerable period the external application of this solution has found favour in the hands of many. It is, however, not only useless for the removal of the swelling, but it does little to mitigate the symptoms that are produced by the ganglion. Possibly the persistent application of a strong solution of iodine may bring about so much inflammation as to cause positive harm. Iodine should therefore be avoided, unless it is employed solely as a *placebo*.

2. Prolonged Pressure.—There is no doubt that in a few instances this method of treatment has resulted in the diminution or complete disappearance of the tumour. It is at the best only an uncertain means of bringing about the cure of the lesion, and an apparent cure may be speedily followed by a refilling of the non-obliterated cyst cavity.

3. Evacuation of the fluid by the bursting of the sac wall by means of a sharp blow, as from the back of a book, or by the pressure of the surgeon's thumbs. This way of treating a ganglion is the one that is probably most frequently used, but there are some weighty objections to its employment. In the first place, it is as a rule merely palliative, the cyst wall healing, and the fluid ere long again distending it. It is a very painful method. The blow struck to rupture the sac may fail to do so owing to the thickness of its wall, or may cause more damage than was intended, including even that of producing a fracture of the bone beneath. On the other hand, it has certain distinct advantages. It is simple, has only a small amount of danger, involves no risk of sepsis, and leaves no scar.

Operative Treatment.—1. Puncture of the sac with a tenotome, and the squeezing of the colloid contents out through the aperture so made. This method may be somewhat more satisfactory than the former in being possibly less often followed by a recurrence. It should be done only after the part has been rendered aseptic, for otherwise most untoward results may ensue, leading in some instances to destruction of the synovial cavities of the adjacent joints and tendon sheaths. Pressure is to be applied by a pad of gauze and firm bandaging after the contents have been evacuated.

2. Removal of the colloid fluid by puncture, and the subsequent injection of the cavity of the cyst with tincture of iodine or other irritating aseptic fluid. This may lead to the obliteration of the cavity by the setting up of an inflammation which will cause the surfaces to become united by plastic exudation. It is not a certain method of cure.

3. Extirpation of the cyst by dissecting it as a whole from the tissues to which it is attached. This is the only surely radical means of removal. It has for its advantages that the whole of the cyst can be dealt with, and that with the assurance that it will never make its appearance again. It is objected to chiefly on account of the scar that will necessarily result, though it must be remembered that this is but a very slight one provided that strict asepsis is maintained. For ganglia in the lower extremity no method of treatment could be more satisfactory, but for those that are found about the wrist, and particularly those on its dorsal surface, it is perhaps not so much in favour. After the recurrence of the swelling when other methods of treatment have in their turn failed, there should be no hesitation in recommending and even urging the radical removal of the cyst by dissection.

Ganglion, Compound Palmar.—Though this term is in common use, the condition is not one that should receive the designation of "ganglion," as it is in reality in the majority of instances a true tuberculous tenosynovitis of the sheaths of the flexor tendons in the palm of the hand.

LITERATURE.—Evans. *International Journal of Medical Sciences*, 1892, vol. ciii. pp. 643-656.—Falkson. *Archiv für klinische Chirurgie*, vol. xxxii. pp. 58-85.—Gosselin. *Bulletin de l'academie de médecine*, séries xvi. No. 7.

Gangrene.

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By gangrene is meant the death of a part; but inasmuch as *caseation* and fatty degeneration are both forms of tissue death, and are not included under this heading, the term must be admitted to be a somewhat lax one.

The older authors did not recognise that many of the phenomena they described were really due to micro-organisms grafted upon the gangrenous part, and in consequence their descriptions have to be very materially altered. It must be remembered that loss of pulsation, loss of temperature, loss of sensation, function, and colour, are the sole signs of the death of a part, and that if this gangrenous portion, after being cleansed, be kept wrapped in an antiseptic dressing, no further change will occur in it. Bearing this in mind, the lengthy descriptions which we have in our older books appear very absurd.

Dry and moist are two forms usually admitted by all authorities.

Dry gangrene is generally met with in old people suffering from atheroma. It is simply death with mummification, and in it the gradual contraction of the calibre of the terminal arteries and the absence of moisture render the tissues a very unsuitable soil for the growth of micro-organisms. Hence septic changes rarely affect the dead part, but the neighbouring living tissues may be the seat of severe septic changes.

The part assumes, instead of a purple dark colour, a tallowy white colour, mottled here and there with brownish streaks due to disintegrated hæmoglobin. The dead tissues become brown, then black, the whole gradually getting mummified.

Moist gangrene is for the most part caused by some obstruction of the venous rather than of the arterial system, so that a complete stasis in the capillaries takes place. If the cause is on the arterial side it is due to *acute* arterial thrombosis, and in the moist, unlike the dry form, there is no previous narrowing of the arteries.

It must be clearly understood that what is generally described as the third stage, or stage of putrefaction, does not exist as part of the gangrenous process, but is due to the presence of micro-organisms in the skin. If the skin be thoroughly disinfected immediately before its death the part will remain aseptic. Thus we recognise two forms of moist gangrene.

(*a*) *Aseptic Moist Gangrene.*—The gangrenous portion, although black, green, or yellowish white, does not change its *size*, and is soon thrown off or absorbed without much disturbance to the surrounding tissues. A good example of this condition is seen in a strangulated aseptic piece of omentum.

(*b*) *Septic or Putrid Moist Gangrene.*—Decomposition is the chief feature in this form of gangrene. The part becomes swollen and boggy, and gives when pressed an emphysematous feeling. Sulphuretted hydrogen, ammonia, and other gases are formed, and give rise to bullæ beneath the cuticle.

The *symptoms* of many cases of gangrene, as described in older works, must be much altered when we recognise that the great majority of the symptoms then described were due really to the absorption of the products of micro-organisms. Practically the symptoms resolve themselves into those produced by a constitutional condition prior to the onset of the gangrene plus the local absorption of ptomaines, or septic poisoning.

These constitutional conditions are most frequently some form of *malnutrition*, *renal disease*, or *diabetes*.

VARIETIES OF GANGRENE

(i.) *Traumatic*, or that due to *injury* of the vessels or tissues. It may be divided into two forms—one *direct*, where, so to speak, the life is crushed out of a part; and *indirect*, where the gangrene occurs at some distance from the seat of the vessel injured. When a part is so severely contused that it is likely soon to be gangrenous, it becomes cold, insensible, and soon its function is interfered with. At the same time there is the change of colour already described. Most forms of direct traumatic gangrene assume the moist characters. They are generally aseptic or become so, because it is so difficult to cleanse the parts thoroughly, and carters and that class are those in whom it is most often seen.

Direct gangrene must not be confused with acute inflammatory or spreading traumatic gangrene, which has special features due to a special micro-organism, but the term must be applied to those cases where so much injury is inflicted on the vessels and tissues that death occurs.

The chief causes of indirect traumatic gangrene are:—(*a*) *Injuries to the main artery and vein.* This is seen frequently in the case of the popliteal or lower femoral arteries, where a compound fracture tears both vein and artery; or it may be that blood is effused to such an extent that it presses on both main artery and main vein and so obstructs the flow of blood within them. (*b*) *Ligature of a main artery* in some people will cause gangrene. (*c*) *Thrombosis* similarly may cause gangrene. If occurring after an injury to a main artery at some distance from where the gangrene exists, it would come under this heading.

(ii.) *Senile gangrene* is one of the commonest forms of gangrene met with. It arises generally from slight injuries to the feet of old people. The calibre of the arteries of the aged becomes very much curtailed from atheroma, and added to this there may be varicose veins or some chronic bronchitis and emphysema. In such a patient anything that upsets the circulation and tends to clotting of blood, either in the large or small arteries, will cause gangrene of toes or foot. The change occurs sometimes in the nose, hands, or tongue, as well as in the lower extremity. The writer's experience is that in the large proportion of cases of senile gangrene there will be found a small abrasion from the boot or from the cutting of a corn, which admits micro-organisms into the small veins, and these easily become thrombosed with a thrombus composed of fibrine and micrococci. This agrees with the descriptions we find of pain, and afterwards a reddish blush, which almost always precede the actual gangrene.

The further history of a case of senile gangrene is, that the patient gets ulcerations, bedsores, a low form of septicæmia, or dies of some complication, such as pneumonia, bronchitis, or exhaustion.

Chemical Changes.—The tissues become oxidised in such a way that carbonic acid, ammonia, and water are the results. Valerianic, butyric, and carbolic acids are formed together with indol, skatol, etc. The gangrenous odour is thus produced. Leucin, tyrosine, margarine, and ammonio-magnesium-phosphate are also formed.

(iii.) *Gangrene due to sudden Obstruction of the Blood-vessels.*—This form may be due to pressure from *without*, as in tight bandaging, pressure of a fracture, or where a plaster of Paris bandage has been badly applied, head of a dislocated bone, or to the application of a ligature. It may also be due to obstruction *within* the vessel (embolic). In the former the limb is at first cold, it is pale in colour, and there is tingling in the peripheral nerves; then the parts get deeper in colour, and soon become purple. Sensation is lost, and the case usually assumes the moist form.

The embolus in the embolic form is usually a fibrinous vegetation, or an atheromatous plate. Neither in the case of embolus nor ligature should gangrene occur if the collateral circulation is good, but there is often present some general condition predisposing to a weak collateral circulation, such as endocarditis in rheumatic fever, or the weak heart of continued fevers.

The pain in *embolic* gangrene is generally felt at that portion of the artery where the embolus has become impacted. This is frequently at the bifurcation of a main artery into its two divisions, as in the popliteal or brachial arteries. A word of advice must be given about the danger of putting severely contused fractures in plaster of Paris, and sending the patients to their homes. This is a practice which is much in use in the German hospitals, but the author has had two patients sent to him by medical men who had done this injudiciously, and gangrene resulted in both cases.

(iv.) *Diabetic Gangrene.*—This comes under the head of gangrene due to general causes. It may be moist or dry, is usually the former, and is due to an endarteritis and peripheral neuritis. The forerunner of grape sugar, or grape sugar itself, causes irritation of the lining of the arterioles, and a thickening and narrowing of their calibre results. Micro-organisms grow very readily in diabetic patients, as is seen in the great tendency diabetics have to form carbuncles.

Diabetic gangrene is as a rule more acute in its course; there is more sloughing and inflammation than in the other forms.

(v.) *Gangrene from ergot* may occur in epidemics, and is due to rye being infected with *claviceps purpurea.* It is preceded often by intestinal symptoms, such as sickness and diarrhœa, and afterwards by coldness and anæsthesia of the limbs. There are two causes of this anæsthesia and coldness—one is contraction of the arterioles, the other a peripheral neuritis. It is considered by some that the neuritis has more to do with the disease than the contraction of the vessels. Most common in men between thirty and forty, the gangrene of ergot generally is of the dry variety, and may be very slight or involve a whole limb.

(vi.) *Symmetrical gangrene* or Raynaud's disease, as its name implies, affects both sides of the body, thus differing from senile gangrene. Again, the calibre of the arteries in this disease is not contracted. The disease essentially is either a peripheral neuritis or some deep-seated lesion of the spinal cord. It has been started by sudden mental shock or fright, and generally occurs in women.

Maurice Raynaud published his thesis in 1862, and his final contribution in 1874. He described a *local syncope*, a *local asphyxia*, and *symmetrical gangrene*, as three successive stages of this disease.

Local Syncope.—Raynaud was inclined to lay stress on the neurotic origin of this disease. Complete pallor of the two hands occurs, or it may be in the toes, one finger or one toe being affected. The principal difficulty is the performance of slight movements, and often no discomfort is complained of. There is analgesia, tactile sense is present, and changes of temperature can be appreciated.

The attack may last a few minutes or some hours. It is brought about by slight changes of temperature, such as a cold day in the middle of one or two warm ones, or it may occur periodically after the bath.

Neurotics and neurasthenics are the classes in whom it is met with most frequently, and there may be a periodicity in the attack, as after the morning bath.

Slighter causes even than this may produce an attack, such as passing from a warm into a colder room. The mild cases do not tend to go on to the later forms of Raynaud's disease.

Local Asphyxia.—Either the whole hand or foot on each side of the body becomes purplish black, or the attack is limited to one or two fingers or one or two toes on each side.

There appears to be nothing but venous blood circulating in the part affected. Some swelling and a burning or shooting pain accompany the attack, which may last for an hour or two, may often recur, and need not necessarily go on to gangrene. The subsidence of the attack may be sudden or gradual. The more severe cycles last from two to ten months. In addition to the fingers and toes which are most commonly affected, patches may occur on other parts, as about the knees or on the helix of the ear.

If the asphyxia persist, then we get the final stage (*symmetrical gangrene*) of Raynaud. The pain in and darkness of the parts increase, and bullæ may form. There appears to be for a time death of a large area, but this settles down to a more limited portion, as the matrix of a nail or end of a finger. The affection may be simultaneous, or successive parts may become gangrenous. The association of Raynaud's disease with *hæmoglobinuria* must not be forgotten, as many cases of the two diseases running side by side have been reported.

The original hypothesis of Raynaud still seems to hold good as the best explanation of the pathology of this disease, viz. "That vaso-motor centre or centres are unduly irritable, that the commonest irritant is from the periphery, as for example cold, and that the efferent impulses from the centre lead to the paroxysmal contraction of arterioles."

(vii.) *Gangrene due to Heat or Cold.*—(*a*) *Frost-bite.*—When a part is exposed to cold the cutaneous capillaries contract, and the part becomes pale and numb, and soon the tissues assume a wax-like appearance. The further results are usually indirect from secondary inflammation.

The symptoms are numbness, tingling, loss of power and sense of weight, generally in the hand or foot. The limb is at first contracted, and shortly afterwards becomes swollen. It is not unusual for parts to look well for some days and then become bluish, dark blue, and finally black. Subsequently a line of demarcation forms, ulceration takes place, and either a toe or a finger, or possibly a foot or hand, is lost. The ulceration may amount simply to a small patch of skin, say about the os calcis or on the outer side of the foot.

Morbid Anatomy.—Cold has at first a stimulating effect upon the circulation, but if it be severe or long continued, then the blood in the smaller veins becomes frozen, and the skin assumes very much the appearance that is seen after the use of the ether spray.

The blood is absolutely frozen and there is complete stasis, and when thawing takes place, so much damage has been done to the vessel-wall itself that it permits the blood to pass through, and hence we get those inflammatory changes which are the real cause of gangrene in frost-bite.

A still longer exposure and the involvement of a greater extent of surface induces the collection of blood in the larger veins, and thus the internal organs become œdematous, and there is fatal stupor, coma, and asphyxia.

The danger from frost-bite is in proportion to the length of time and extent of surface exposed, and to the power of resistance of the individual. It must be remembered that alcoholics are very liable to frost-bite.

Dry cold is said to have a more severe effect than moist cold, and if a wind is blowing the effect will be much more severe. The author had under his care seventeen patients from a crew shipwrecked on the Newfoundland coast. An important observation was made by the men, namely, that those who had not removed the water out of their sea-boots had escaped, while all those who had became affected with gangrene. They were in the open for sixteen days, and the presumption was that the water was heated by the body and kept the feet from freezing.

Cases are related where persons have been buried in *dry* snow for many days without much food and recovery has taken place, but if the snow has been thawing, a very much more serious effect has been produced.

This is singularly well pointed out by Larrey, the distinguished French surgeon, who, in the Russian campaign, found that though none of the soldiers suffered from many days' exposure to *dry* cold and frost, as soon as a thaw came a large number of both guard and line became frost-bitten.

Chilblain is a less serious local effect of cold, as are chaps, fissures, and frost erythema (pernio). The lips, the ears, the fingers, and toes are the portions of the body most frequently affected. Young girls of lymphatic constitution or those who inherit an irritable nervous system are most liable to this complaint.

Sudden changes of temperature are generally attributed as causes, but many people get chilblains who never leave the house in cold weather. As in frost-bite, so in this disease, damp cold, as in a sudden thaw, is a much more frequent exciting cause than dry cold.

An eruption of dull red patches, not well defined, raised, tumefied, and burning, may be followed by vesicles or bullæ. Subsequently an ulcer excavated and with thick edges may form. A peculiar viscid discharge is often present. Three degrees have been described: (1) simple congestion with itching and extreme tenderness; (2) vesication; and (3) death or sloughing with or without marked erythema.

Chilblain is merely an early stage of frost-bite, according to some authorities, but there is little doubt that in most cases of chilblain the nervous system, as in Raynaud's disease, plays an important part.

(*b*) *Burns.*—It is in the last three degrees of burns and scalds that the effects produce gangrene. In these three we find that sloughs have to be removed before the healing process can take place. These effects will depend on the source, duration, and intensity of the heat.

(viii.) *Gangrene due to the use of carbolic acid* is met with under dressings of this acid. It is of the dry variety and affects the fingers and toes for the most part. Apparently it is more often met with after the prolonged application of weak solutions, and is not so often seen after the application of *pure* carbolic acid.

A weak terminal circulation should contra-indicate the use of carbolic acid dressings. The American authorities speak of as many as one case of carbolic gangrene in 1000 surgical cases, but this is certainly not the experience in this country. Personally I have seen only one case of gangrene that could be certainly traced to the use of carbolic acid.

Multiple gangrene of the skin occurs independent of the use of carbolic acid. Blebs occur in many parts, and afterwards numerous small particles of skin die. The nervous system is supposed to play an important part in this disease.

(ix.) *Infective gangrene*, or the gangrene due to the presence of micro-organisms.—This is a most important heading, and one which includes such diseases as glanders, plague, beri-beri, hospital and acute spreading traumatic gangrene, etc.

(*a*) *Spreading traumatic or acute traumatic gangrene* is an extremely rapid and very often fatal form, due to a micro-organism, the bacillus of malignant œdema, first isolated by Koch. It is closely allied to symptomatic anthrax in cattle. The bacillus of malignant œdema is rod-shaped, somewhat like anthrax, but more slender. It is capable, even in a test-tube, of forming hydrogen and carburetted hydrogen. It grows very readily in musk, and is found in garden earth.

The course of the disease is very rapid. It, as a rule, appears from two to three days after a severe accident, generally a compound fracture or a compound dislocation, but it may occur after a small inoculation. It is usually fatal in three or four days if very active measures are not adopted. As a general rule it is predisposed to by drunkenness which has been long continued, as for instance in barmen and brewers' draymen.

The limb becomes hard, tense, white, and œdematous, but soon goes on to a dusky red and then livid colour. There is gas, chiefly hydrogen and carburetted hydrogen, evolved, and crackling is present; at the same time the constitutional symptoms are very severe, and delirium soon supervenes.

(*b*) *Hospital gangrene* and wound phagedæna are now very rarely seen. I have never seen a case, but before hospital wards were so well ventilated and antiseptic precautions taken it was a common enough disease. A pseudo-membranous slough forms within eight hours to three or four days of the infliction of a wound; the granulations, if present, become ashy gray. The surface of the wound is dry and the edges pale. Then either an ulcerative or gangrenous process follows. In the gangrenous form the disease is often fatal in from twenty-four to forty-eight hours.

(*c*) *Cancrum oris or Noma.*—Under this heading we have to deal with a very fatal disease which occurs in children from 2 to 7 years of age, and in most cases after measles or scarlatina, especially the former. On the gum or immediately inside the cheek an ulcer occurs, which at first is slimy and white, but soon becomes gangrenous; a black spot appears on the outside of the cheek and rapidly spreads, so that the side of the face may be destroyed, the bones occasionally being involved. In other cases the process is a gangrenous stomatitis, giving rise to a foul breath from fœtid material which has been swallowed. The symptoms of acute poisoning (sapræmia) soon show themselves, and the patient becomes comatose and dies in three or four days. Septic pneumonia is often a cause of death in this disease. (See "Stomatitis.")

Bacteriologically a long delicate bacillus has been proved to be the cause. The author has had under his care two cases of noma which recovered after the free use of the thermo-cautery, and in which extensive plastic operations had subsequently to be performed. The word noma is sometimes restricted to this process occurring about the genitals of children.

(*d*) *Carbuncle and boil and acute necrosis* are classed by some authors as forms of gangrene.

(*e*) *Gangrene in beri-beri* has been seen. A thin black line crossed the tips of the toes, and dry gangrene followed in both feet, requiring amputation of both legs at the seat of election. Undoubtedly the gangrene was due to a peripheral neuritis which so often occurs in this disease. The neuritis is caused by the direct action of toxins (produced by a micro-organism) on the nerve itself.

TREATMENT.—Some general remarks will be made on the treatment of gangrene, and then the treatment of each variety will be dealt with shortly.

To begin with, the whole treatment of gangrene must be altered now, our methods have so much improved, and especially is this true as regards the question of amputation. We can, with safety and with much less chance of sloughing of flaps, amputate very much earlier than we formerly could, thus saving the patient much painful suffering and danger from the presence of a septic dead mass of tissue. A second important result of our better antiseptic methods is the fact that we can boldly cut down on a ruptured aneurysm or compound fracture, relieve pressure on vessels of effused blood, and thus often prevent gangrene. It is most important in all cases of gangrene to get at the exact cause; thus a tight bandage should be removed at once, or a free incision should be made in inflammatory tissue, if causing obstruction to the flow in the main vessels. The stricture in a piece of strangulated bowel or omentum should be divided at once, and so with a paraphymosis, if elevation and attempts at reduction fail.

If in a limb the main vessels cannot be relieved, then the limb should be raised, the joints flexed, and the part should be lightly wrapped in cotton wool.

If the gangrenous part has not become septic, then the greatest care must be taken to cleanse skin, nails, etc., and salicylic wool and iodoform should be placed between the toes or similar situations after thorough cleansing. If septic, such a limb should not be poulticed, but a boracic fomentation should be placed a little above the line of demarcation.

Sloughs have to be removed, and the best application is a bran poultice covered with boracic powder.

Wait for a line of demarcation, keep aseptic if possible, but don't wait too long, especially in septic or spreading cases, are the broad rules which must guide our judgment.

A nourishing diet, stimulants, and tonics are also required, and small doses of opium, except where there is diabetes or renal disease.

Where these constitutional diseases exist, special dietetic and medicinal treatment must be employed.

Traumatic.—Warm antiseptic lotions must be used freely to thoroughly cleanse. Ascertain if there is any pulsation; if not, see that the main vessels are not pressed on by extravasated blood or compound fracture. Do not amputate at once if there is much shock, but rather wait for eight or ten hours. I quite agree, from a large experience of severe smashes, with those authorities who advocate waiting in order to let the patient recover and the parts be made aseptic. The rule for amputating is, to do so, only when you are convinced that there is no circulating blood in the tissues.

On the other hand, we must not try to save the part in the case of albuminuria, diabetes, or other constitutional disease.

The subject of these diseases will not stand the long-continued drain upon them, and very often amputation has to be resorted to later, when strength to rally no longer remains.

Raynaud's Disease.—Preventive, tonics, frictions, warm douches, electric baths, attend to any uterine complication, keep aseptic. In no case is it necessary to amputate. Rather cover with iodoform and allow tips of fingers to gradually drop off.

Ergot.—In this form it is very rare to find more than a finger or toe affected, and amputation is not generally necessary.

Diabetic.—Codeine ¼ to 5 grs., or small doses of opium, very useful. In such cases any septic wound should be specially looked after, as its neglect is highly dangerous.

Amputate very early; rule here completely changed from what it used to be. If there is a plug high in a main vessel, don't do a regular amputation, but dust with iodoform, divide bone, and allow to drop off gradually.

Senile Gangrene.—Early amputation is now the treatment, well away from the gangrene, and in the case of the foot through the knee-joint, even if pulsation be felt below. The patient here must be warned about septic abrasions and wounds about the toes.

Frost-bite.—Prevent, by warm clothes and absence of moisture. Patient should eat much fat and avoid alcohol. When actually frozen, the part should be most carefully thawed with cold or snow water and gentle friction. Gradually raise temperature around patient. Elevate if œdema.

Render, if gangrenous, aseptic with dry applications. Amputate early. For the milder forms, as in chilblain, the same treatment must be adopted, and painting with fairly strong solutions of carbolic acid, or belladonna and collodion, or the old-fashioned brandy and salt mixture, are amongst the best remedies.

Acute spreading Gangrene.—The point in this disease is to remember not to wait for a line of demarcation, but to amputate as soon as possible. The shoulder or hip operation must be chosen, and care must be taken not to soil the flaps. Stimulants, incisions, and baths will be necessary. It is a most fatal disease; about 5 per cent recover.

Noma.—If in mouth, antiseptic washes, sanitas, and boro-glycerine. Then scrape with Volkmann's spoon, or destroy with actual cautery or pure carbolic acid, remove dead bone. If recovery take place, Thiersch-Grafting or plastic operations will be required. Iron, quinine, and stimulants will be necessary.

If in genitals, then baths of sanitas or boracic acid, and powder with iodoform.

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Gas Poisoning. *See* TOXICOLOGY.

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CONGENITAL ANOMALIES

I. *The Œsophagus.*—Congenital anomalies occur more rarely here than at any other part of the alimentary tract. The following conditions have been found:—

(1) Transposition.—In association with transposition of the stomach the œsophagus passes down on the right side of the vertebræ.

(2) Absence or Obliteration, partial or complete.—One variety of this shows very constant characteristics: the pharynx with the upper ¾ inch of œsophagus is dilated, and forms a blind pouch ending just below the level of the cricoid cartilage; below this the upper part of the œsophagus is absent or represented only by a band of fibrous tissue, or a few muscular fibres; the lower part is normal, and opens normally at the cardiac orifice, but above it communicates, by an opening about $\frac{1}{10}$ inch in diameter, with the trachea ½-¾ inch above the bifurcation. Such a condition may be suspected during life when all food is regurgitated through the nose and mouth almost directly it is taken, and a sound cannot be passed down the pharynx more than about 5 inches from the lips. Death occurs from starvation in a few days; one case at Great Ormond Street lived fourteen days with rectal feeding.

The most satisfactory explanation of this condition is that suggested by Mr. Shattock (*Path. Soc. Trans.* 1890), who assumes that at the time of the budding of the diverticulum from the anterior wall of the mesenteron to form the lower part of the trachea and the lungs, a kinking forward of the posterior wall occurs, so that the lumen of the mesenteron, which should persist as the œsophagus, is occluded just above the level where the tracheal diverticulum is given off. The segment of the mesenteron below the kink, subsequently the lower part of the œsophagus, remains in communication with the tracheal diverticulum. "On the subsequent formation of the upper portion of the trachea the whole of the unduly contracted lumen (of the mesenteron) immediately above the diverticulum becomes involved in the formation of the air-passage."

(3) Stenosis.—This occurs most often at the upper part of the œsophagus about the level of the lower margin of the cricoid cartilage, more rarely at the lower end just above the cardiac orifice. This condition may be recognisable during life by the difficulty in swallowing. It has been treated by the passage of bougies.

(4) Diverticula.—It seems doubtful whether any diverticula from the œsophagus are actually congenital. A shallow pit is sometimes found in quite young children at the level of the bifurcation gland, where this has been caseous, but this is probably the result of traction rather than a congenital anomaly. The congenital diverticula which result from persistence of branchial clefts communicate with the pharynx, not with the œsophagus.

II. *The Stomach.*—(1) Abnormal Position.—The stomach may be transposed together with the heart and other viscera. It may project alone, or with part of the transverse colon or small intestine, into either pleural cavity, usually the left, owing to some deficiency of the diaphragm.

(2) Persistence of Fœtal Shape and Position.—The tubular shape and vertical position are sometimes retained. Some authors state that in early infancy some tendency to such a fœtal condition is the rule, and no doubt this is true of the vertical position, but in the writer's experience any tendency to tubular shape has been extremely rare.

(3) Abnormal smallness has occasionally been observed.

(4) Hour-glass contraction or a partial septum may almost divide the stomach into two compartments.

(5) Stenosis or atresia of the cardiac, or less rarely of the pyloric orifice, may be present, and is probably quite distinct from the congenital hypertrophy of the pylorus described below.

III. *The Intestines.*—(1) Abnormalities of position are very common in the large intestine apart from transposition of viscera which may involve the intestines. The sigmoid flexure, relatively much longer in the infant than in the adult, is often much above its average length even for an infant, and may reach over to the right iliac fossa, or form a loop upwards reaching to the right or left costal margin before turning down to the rectum (*vide* Constipation, p. 111). The cæcum also occasionally lies in an abnormal position, *e.g.* in the mid line at the level of the umbilicus.

(2) Absence or undue Shortness.—This is very rare. In one case the small intestine measured only 2 feet (*Bart. Mus. Cat.*); in another the colon was absent; in another there was no vermiform appendix.

(3) Stenosis or Obliteration.—This is found occasionally in any part of the intestine, but much more often in the small than in the large bowel. Of cases occurring in the small intestine, 42 per cent are in the duodenum (Silbermann). Where multiple stenoses occur it seems likely that a volvulus during intra-uterine life may have caused them. In some cases fœtal peritonitis has been present, and seems a more likely cause. The bowel above the stenosis is usually much dilated, while that below is small, or for some distance may be represented only by a fibrous cord. The condition can only be suspected during life from symptoms of intestinal obstruction, where these are not due to imperforate rectum or anus. Complete occlusion generally proves fatal in a few days; cases of slight stenosis have lived to adult life.

(4) Diverticula.—The persistence of part of the omphalo-mesenteric duct not uncommonly causes a diverticulum in the ileum 2-3½ feet above the ileo-cæcal valve, usually as a blind pouch ½-3 inches in length. The writer found such a diverticulum eleven times in 500 autopsies. Sometimes from the end of the diverticulum a fibrous cord, representing the obliterated portion of the duct, passes to be attached at the umbilicus, or the whole duct may be patent up to the umbilicus, forming a fæcal fistula from which prolapsed mucous membrane often protrudes. In either case the persistent duct serves as a fixed band under or over which coils of intestine may at any time become strangulated. Fæcal impaction may also cause inflammation in the blind diverticulum, and not very rarely the diverticulum is found forming part of an external hernia. Diverticula elsewhere are very rare, especially in the large intestine.

(5) Imperforate Anus or Atresia Recti.—This is due to imperfect development of the hind gut and its failure to meet the proctodæal invagination. It occurs in various degrees: (*a*) there may be only a thin septum separating a well-marked anal depression from the blind end of the gut; (*b*) the rectum may end blindly 2-3 inches away from the surface, and there may be little or no trace of an anal depression; (*c*) there may be a septum occluding the rectum 1-3 inches above the anus, although below the occlusion the rectum and anus are normal; (*d*) the rectum may open into the urethra, bladder, vagina, or even in the loin (*vide* "Diseases of the Rectum").

(6) Rectal or Anal Stenosis.—Without being actually imperforate the anal opening is sometimes abnormally small (p. 113). In such cases it may be stretched under an anæsthetic or enlarged by incision. Similarly the rectum may show narrowing of its lumen or a partial septum at any part of its course, and dilatation or incision may be necessary.

Diseases of the Œsophagus

Acute œsophagitis, as a primary condition, is occasionally found in early life, chiefly during the first year. It is scarcely recognisable clinically. The symptoms are unwillingness to suck, pain on attempting to swallow, and regurgitation of food almost directly it is taken. Tenderness on pressure over the lower part of the trachea is also said to be present (Morell Mackenzie). As a secondary condition œsophagitis has been found with the specific fevers, and the writer has met with it in fatal cases of infantile diarrhœa. In such cases it produces no special symptoms. The application of leeches or of hot fomentations to the neck has been recommended in the rare cases where the condition is suspected during life.

Thrush sometimes spreads down from the pharynx into the œsophagus, and forms a milky-white layer which is found to consist of the spores and mycelium of the so-called "oidium albicans" mixed with epithelial cells; the fungus may penetrate deeply between the cells of the mucosa, and has been found forming so thick a layer as even to obstruct the passage of food.

Diphtheria not very rarely causes a membranous œsophagitis. In forty consecutive autopsies on children with diphtheria the writer found membrane in the lower 1-2 inches of the œsophagus in three cases, and intense congestion of the same area in two others.

Ulceration of the œsophagus is very rare. Superficial erosions have been found as part of the acute œsophagitis of infants, and rarely in older children. The pocks of variola have been found in the œsophagus in children as in adults (Steffen). The least rare variety is tuberculous ulceration from without; the caseous bifurcation gland ulcerates through into the œsophagus about two inches above the cardiac orifice. The writer found such a perforation four times in 270 autopsies on tuberculous children. In none of these cases were any symptoms produced, but hæmatemesis and melæna have occurred. Tuberculous ulceration apart from such direct extension is extremely rare. The writer met with it twice in the same series of cases.

Softening of the œsophagus, like the similar condition in the stomach, with which indeed it is usually associated, is probably an entirely post-mortem phenomenon. It seems to be much commoner in infancy and early childhood than in adults, and it is remarkable that it is much more often found with intracranial disease than with any other condition: the writer met with it eight times in 500 autopsies in children; seven were cases of tubercular meningitis, the remaining one was a meningocele. The lower end of the œsophagus, about 1 inch above the cardiac opening, is almost diffluent, and in the middle of the softened area there is generally a perforation with thin, frayed edges. The connective tissue of the posterior mediastinum has disappeared, and the nerves and vessels are cleanly dissected out by the action of the digestive fluid which has escaped through the perforation. Usually the adjoining parietal pleura on one or both sides is also softened and perforated by digestion, and an ounce or more of opaque reddish brown fluid, sometimes with partially digested food, is found in the pleural cavity. The posterior and lower part of the lung also is softened, greenish brown, and friable.

Diseases of the Stomach and Intestine

Congenital Hypertrophy of the Pylorus.—Since the year 1841, when this condition was first recorded, a considerable number of cases have been observed in which obstruction of the pylorus existed in infants soon after birth, and it seems likely that this condition is less rare than was at first supposed.

Boys seem to be rather more often affected than girls: out of nineteen cases twelve were males, seven females; but the numbers are at present too small to afford reliable information on this point. No family predisposition has been observed.

Symptoms.—The infant is born healthy, and may be a fine child at birth. For the first week or two it takes food well, and there is no vomiting, or only such slight regurgitation as may be natural at this age. This latent period of apparent health varies, in very few cases vomiting has begun on the day of birth, in about half the cases the onset of vomiting has occurred within the first fortnight, in several cases it has been delayed until the fourth, fifth, or even sixth week. Vomiting is the first symptom, and for a time gives rise to but little anxiety, for at this stage it may occur only once or twice in the day. At the same time the bowels are costive. A change of feeding is tried, and the vomiting perhaps diminishes, but after a few days increases again; other changes of diet are tried, but soon cease to have any influence on the vomiting, which becomes more and more frequent until almost every food is vomited. In some cases, especially in the early stage, and when small feeds are given, the food is retained for some hours, and then vomited in large quantity; more often, however, the vomiting occurs a few minutes after a meal, and in the later stage immediately after swallowing. There is usually no retching, and when the condition is advanced the food seems to be pumped up forcibly by the hypertrophied stomach.

The vomit consists of curdled milk or of whatever food has been given, but soon some gastric catarrh occurs, and mucus is mixed with the vomit, and sometimes even streaks of blood. Where there is much dilatation of the stomach the vomit has a frothy appearance. The absence of bile from the vomit is particularly noticeable, as pointing to obstruction of the pylorus. Pain is often absent throughout, but in some cases there seems to be some pain accompanying the act of vomiting.

A very characteristic sign of congenital hypertrophy of the pylorus is visible peristalsis of the stomach; in some cases it is so obvious that it has attracted the notice of the parents, in others it is only noticeable with care, and to elicit it gentle flicking of the abdominal wall may be required. A rounded prominence appears bulging forward in the epigastrium, first just below the left costal margin, and then travelling with a slow vermicular movement across the epigastrium to disappear at the right costal margin. This movement from left to right serves to distinguish it from peristalsis of the colon in which the movement is from right to left.

An even more important sign of this condition is a palpable tumour in the position of the pylorus. This has been present in several of the cases, but has not been detected usually until several weeks after the onset of vomiting; the earliest date at which it has been felt is the twenty-seventh day after birth (J. Thomson), about three weeks after the onset of vomiting. Even in cases where the pylorus is easily palpable, it is not so at all times alike; its palpability depends on the temporary condition of the muscle, and, like an intussusception, it may be felt only when there is some contraction of its muscular coat occurring. For this reason one examination is not sufficient to exclude the presence of a palpable enlargement of the pylorus.

Associated with the vomiting there is almost always much constipation, and when the vomiting becomes severe there is marked diminution in the amount of urine passed. As less and less of the food is retained, wasting becomes extreme, the temperature becomes subnormal, and in most cases the child dies of exhaustion.

Morbid Anatomy and Pathology.—The hypertrophy of the pylorus is due to an increase in the thickness of its muscular coat, chiefly in the circular layer. In a few cases some increase also of the connective tissue has been found, but this is exceptional. The cause of the muscular hypertrophy or hyperplasia is still undetermined; it seems certain that it is partly an extra-uterine occurrence, although there can be little doubt that some congenital abnormality underlies it. Some would regard the excess of muscle tissue as a developmental error, a congenital malformation; others consider that this excess is simply the manifestation of increased function from some cause, either extra or intra-uterine. Perhaps the most probable theory is that put forward by Dr. John Thomson, who suggests that some functional disturbance of the nervous mechanism of the stomach occurs *in utero*, causing antagonistic action of the muscles of the stomach with spasmodic contraction, and so hypertrophy of the pylorus. As a result of the obstruction at the pylorus the muscular wall of the stomach, and sometimes also of the œsophagus, shows considerable hypertrophy, and sometimes there is considerable dilatation of the stomach.

Diagnosis.—A history of persistent vomiting associated with constipation in an infant under six months of age should always arouse a suspicion that there may be congenital hypertrophy of the pylorus, and where the vomiting resists all treatment, and is associated with visible peristalsis of the stomach, and a palpable tumour in the position of the pylorus, the diagnosis is certain. Before these signs have become evident the condition may be mistaken for simple gastric catarrh; this, however, is more often associated with diarrhœa, and the vomiting can usually be referred to some obvious fault in diet, and is more influenced by treatment. It must be remembered that regurgitation of food is quite common and physiological in healthy infants during the first few weeks of life, any overdistension of the stomach being thus provided against.

From congenital stenosis of the pylorus without hypertrophy, diagnosis is impossible unless a palpable enlargement of the pylorus is detected. Stenosis of the upper part of the duodenum may also closely simulate this condition.

Prognosis.—There is increasing evidence to show that congenital hypertrophy of the pylorus is not always fatal, and in one case which recovered and died nearly a year later of broncho-pneumonia, the hypertrophy of the pylorus could still be demonstrated (F. E. Batten). In most cases, however, it proves fatal. In nineteen cases in which the age at death was recorded, the average age was 9½ weeks at death, the earliest was twenty-one days, the latest nearly five months. The interval between the onset of symptoms and the fatal ending varies from eighteen days up to twelve weeks.

Treatment.—The most valuable method of treatment is feeding by stomach-tube through the nose or mouth. In several cases this has produced considerable temporary improvement, and in one case a spontaneous recovery seemed to follow the improvement started by nasal feeding. Drugs are of some value when gastric catarrh is present as a complication; in such cases sodium bicarbonate, with a minute dose of aqua laurocerasi, may be useful, or the stomach may be washed out with a weak alkaline solution. Where dilatation is considerable and the food collects in the stomach, this should be washed out daily. Careful dieting must not be neglected, for the vomiting is certainly influenced to some extent by the food; it is important, therefore, to select such food as is likely to prove least irritating to the stomach; for this purpose it may be wise to peptonise the milk, or to use whey with or without cream (*vide* "Infant Feeding").

The question of surgical treatment naturally arises, but at present there is not sufficient experience to show how far it may be of value. It has been tried at least three times—twice with rapidly fatal result, once with a favourable result at the age of eight weeks (Abel). It is obvious that such a severe operation as gastroenterostomy, or any of the procedures which would be necessary to relieve the obstruction, cannot be otherwise than extremely risky at so tender an age and in so feeble a state. For the diagnosis can hardly be made with certainty until some weeks have elapsed since the onset of vomiting, and by that time the infant is in poor condition for such an operation. Moreover, as it seems now quite certain that cases have recovered without operation, it may be said without hesitation that the risk of surgical interference should never be undertaken until a fair trial has been given to medical treatment, especially to the method of feeding by stomach-tube.

Gastritis.—Gastritis is an occasional result of acute dyspepsia in infancy. As the result of irritant poisons it may, of course, occur at any age.

Membranous gastritis is found occasionally in children, usually with diphtheria. The membrane is situated near the cardiac orifice, and is associated sometimes with membrane in the lower part of the œsophagus, and almost always with severe pharyngeal diphtheria. The writer has also found membranous gastritis associated with membranous colitis in an infant with severe broncho-pneumonia where there was no evidence of diphtheria. It has also been seen in tuberculosis.

The condition cannot be recognised during life, but has been associated usually with severe vomiting.

Gastric ulcer is very rare in childhood. It occurs most often in new-born infants probably as a result of congestion owing to the circulatory changes at birth. It is then sometimes solitary, but perhaps more often occurs as multiple erosions. Such ulceration either in the stomach or in the duodenum may give rise to fatal hæmatemesis and melæna; these symptoms occur almost always before the tenth day, usually within the first forty-eight hours of life (*vide* "Diseases of the New-born").

Follicular erosions have been found in severe cases of gastric catarrh in infancy, sometimes also in association with specific fevers, and sometimes with severe burns (S. Fenwick). They very rarely give rise to any special symptoms; severe hæmorrhage has, however, occurred. Perforating gastric ulcer, exactly similar to that found in adults, has occasionally been found in children; the earliest case was at 2½ years.

Tuberculous ulceration of the stomach is less rare in infancy and early childhood than at any other period. The writer met with it six times in 270 autopsies on tuberculous children; in three of these the tuberculous character was verified by microscopic examination. In all the recorded cases there has been extensive tuberculosis in other parts of the body, often in the intestine and peritoneum. Special symptoms are usually lacking, but fatal hæmatemesis has been recorded (Bignon), and also perforation with acute peritonitis (Cazin).

Hæmatemesis is very rare in infancy. Apart from its association with melæna neonatorum and the other causes already mentioned, it is seen occasionally in breast-fed infants in perfect health. On examination it is found that the mother's nipples are cracked and bleeding, and it is evident that the infant has sucked the blood from the nipple, and hence this spurious hæmatemesis.

Malignant Growth.—Only a few cases in infancy and in early childhood have been recorded. Cylindrical-celled epithelioma at the pyloric end of the stomach was found in an infant aged fifteen weeks (Cullingworth). Sarcoma of the stomach at the age of 3½ years has been recorded (Finlayson); the writer found secondary sarcoma of the stomach in an infant aged six months. Lymphadenomatous growth in the stomach has been seen at the age of eighteen months (Rolleston and Latham).

Softening of the stomach (gastro-malacia), like the similar but less common condition in the œsophagus, was at one time thought to be an ante-mortem phenomenon, it is now almost certain that it is entirely the result of post-mortem digestion. It appears to be commoner in children than in adults. The softening is generally at the cardiac end of the stomach, which is much thinned, and has a greenish brown colour, while on the inner surface the black arborescent vessels of the mucosa are seen; occasionally a ragged perforation with thinned, almost diffluent edges occurs, and partially digested food is found in the peritoneal cavity.

Disorders of Digestion

The disorders of digestion may perhaps be considered most usefully under the headings of their prominent symptoms. Such a method may seem less scientific than the use of a pathological classification, but for the clinician, at least, it has the advantage of corresponding with what is certain rather than with what is often quite a matter of speculation. In a very large number of the cases of gastro-intestinal disorder in infancy it is impossible to determine whether the disturbance is functional, catarrhal, or even associated with such gross lesions as ulceration or membranous inflammation. While, therefore, it must be recognised that there are such pathological differences, and that in some cases they are recognisable clinically, the subject will be considered here mainly from a clinical standpoint.

Flatulence and Colic.—These two conditions are closely associated and are extremely common in infancy. The accumulation of flatus in the stomach or in the intestine is almost always the result of improper feeding; it may be that one or other constituent—casein, starch, or sugar—is in excess, or the food may be given in too large quantities or at too short intervals. Flatulence may be due to air drawn into the stomach by sucking at an empty bottle, or at a perforated teat used as a "comforter." But sometimes no cause whatever can be found; a child fed at the breast with the utmost care may suffer from flatulence and colic, and that too when the breast milk appears to be good. In such cases one can only suspect that the milk may not be so good as it looks; it may contain too much fat, or its composition may be affected by some disturbance of the mother's health; sometimes it is evidently thin and watery.

Colic sometimes occurs where there is no flatulence. Coldness of the extremities and insufficient covering for the abdomen seem responsible for it in some cases; the presence of undigested food, and especially of masses of constipated fæces in the intestine, causes it in others. Occasionally it may be due to the passage along the ureter of small uric acid concretions, such as are found not very rarely in the pelvis of the kidney in infants.

Symptoms.—Colic in an infant is easily recognised. The child screams during the attack and draws up its legs, the muscles of the abdominal wall being hard and contracted at the time. This may occur at intervals for an hour or more, and then perhaps after the passage of flatus from the mouth or bowel the child becomes easier; or the screaming may continue until from sheer exhaustion the child becomes quiet and falls asleep. Such attacks are very apt to occur in the evening, and the child may be quite free from them during the day. When the colic is severe, other symptoms are often noticed; the infant becomes pale or livid gray about the lips, and there is often slight twitching of the lips, or the eyes are rolled upwards, or the limbs become rigid and the fists clenched. This convulsive tendency may go further, and general convulsions occur. A weakly infant after an attack of colic sometimes becomes quite pale and alarmingly collapsed.

Diagnosis.—It is not always easy to determine the cause of colic in infants. In a large number of the cases some indication of the cause is found in associated symptoms of chronic dyspepsia or gastro-intestinal catarrh, such as chronic vomiting or diarrhœa and marasmus; sometimes the sour smell of the eructations points to fermentation in the stomach; often the relation to meals is obvious, each attack occurring soon after the infant has been fed. It is always wise to examine the abdomen in these cases; the flatulent distension of the stomach or the bowel may be distinctly seen and felt, or scybala may be detected in the intestine. The possibility of intussusception must also be remembered.

Treatment.—In a breast-fed infant the frequency and quantity of the feeds must be regulated, and any defect in the mother's health must be treated. It is hardly ever necessary to wean altogether, but if the milk seem to be poor it may be advisable to substitute hand-feeding two or three times a day, continuing suckling at other times. In hand-fed infants the diet must be carefully revised (see "Infant Feeding"). Usually the casein must be diminished or the curd made more digestible by dilution with lime water, oatmeal water, or barley water. Some of these infants are suffering from the results of a diet which contains much starch, especially some of the patent "Foods"; starch, in general, is to be avoided. Where there is evidence of fermentation in the stomach, stomach-washing may be useful; and if the stools are offensive, gray powder with soda is likely to be effectual. The presence of scybala requires the use of an aperient enema and subsequent laxative treatment. A teaspoonful of dill water given just before a feed or mixed with it, or some carminative mixture, such as Tr. card. co. or Spir. ammon. aromat., with bicarbonate of soda and some aromatic water, sometimes prevents the attacks. When the flatulence appears to be chiefly intestinal, creasote (♏ $\frac{1}{4}$-$\frac{1}{2}$) or resorcin (gr. ij.-iv.) may be given three or four times a day. In all cases it is important that the abdomen and thighs should be warmly covered.

During the attack hot flannels should be applied to the abdomen, the feet should be warmed by putting them in hot water if necessary, and the child's position should be shifted frequently to encourage the passage of flatus. This may be assisted by friction or pressure over the abdomen, and the relief afforded in many cases by the prone position as the child lies over the nurse's knee or shoulder, is no doubt due partly to the pressure thus applied. A teaspoonful of hot water with a few drops of brandy or a little aqua chloroformi may give relief, and sometimes a small teaspoonful of salad oil will stop the pain. An enema of soap and water should be given at once if scybala are present, and in any case an enema of plain warm water may do good. If necessary a drop or two of laudanum may be added to the enema, but the use of opiates, particularly in the form of patent "soothing powders" or "syrups," is, as a rule, strongly to be discouraged, although under medical supervision minute doses of Pulv. ipecac. co. (gr. ¼-½ for an infant of four to nine months) are sometimes advisable for a few days, especially where the attacks are followed by much prostration.

HICCOUGH is frequently troublesome in infants, especially during the first few months of life. It is associated usually with some digestive disturbance, particularly with the condition of flatulence described above. Often, however, no such cause can be found, and there is nothing to account for the onset of this spasmodic action of the diaphragm.

The attacks of hiccough in some cases occur almost daily, and although not serious, are sufficiently distressing both to the infant and to its parents to require treatment. When the attack is prolonged a weakly infant is sometimes quite exhausted by it, and even in milder degrees the discomfort it causes to the child is often shown by a fretful cry as each spasm occurs.

Treatment.—When the attacks are frequent the diet should be carefully considered; it may be that some slight alteration, such as has been recommended in the cases of flatulence, may prevent the hiccough. The attack may be shortened sometimes by very simple methods, such as changing the baby's position at short intervals, gentle patting on the back, or friction over the upper part of the abdomen. In other cases a teaspoonful of hot water, either alone or with one or two drops of sal volatile, or five or ten drops of brandy, may be successful. The administration of a weak acid, such as lemon juice, or one or two drops of dilute sulphuric acid in a teaspoonful of cold water, is sometimes very effectual.

VOMITING.—Under this head must be mentioned first the regurgitation of food, which is so common during the first two or three months of infancy as to be almost physiological; it might perhaps be distinguished as *repletion-vomiting*. This is seen in infants who take their food greedily whether at the breast or from the bottle. A few minutes after, sometimes even before the meal is finished, they regurgitate without effort and without discomfort a small part of the contents of the stomach. This vomiting seems to be really a protective arrangement to prevent over-distension of the small infantile stomach. As a rule it has no ill effect, and the child gains weight steadily in spite of the vomiting. Nevertheless it is wise to check this regurgitation by reducing the quantity of each meal, for the continual over-feeding is apt to lead to actual gastric catarrh, and sometimes certainly, as Dr. Goodhart suggests, it seems as if a habit of vomiting were established which it is difficult to break.

Apart from such repletion-vomiting, the commonest cause of vomiting in infancy is some form of gastric irritation. In many cases the feeding is faulty, either as to quantity, quality, or frequency of food. Sometimes the irritation appears to be toxic, as where vomiting is due to sour milk or bad eggs, and it is probable that the vomiting which occurs in severe cases of summer diarrhœa is sometimes, at least, toxic in origin. Vomiting is very frequently associated with diarrhœa, indeed it is comparatively seldom that an acute attack of vomiting occurs in an infant without some intestinal disturbance. Vomiting in infancy is rarely obstructive; the possibility of some congenital narrowing of the pylorus or duodenum, however, must be borne in mind in any case of persistent vomiting in early infancy (*vide* Congenital Hypertrophy of the Pylorus): intussusception also, strangulated hernia, and the rarer forms of acute intestinal obstruction must not be forgotten.

Apart from disorders of the gastro-intestinal tract, vomiting is a common symptom of the onset of many acute diseases in infancy; it may also be due to some intracranial disease, meningitis or tumour.

An *acute* attack of vomiting in an infant is perhaps most often the result of acute dyspepsia. The infant seems ailing; it lies quiet and pale, or cries with colicky pains. The temperature may be raised to 101° or 102°. Vomiting occurs at short intervals, and lasts a few hours, ceasing when the stomach is emptied. There is usually some slight transitory diarrhœa. This begins soon after the vomiting has commenced, and often lasts a day or two after the vomiting has ceased. This may constitute the whole illness in a mild attack, but in more severe cases, or where the irritation is increased by the continuance of improper food, the vomiting may continue even after the food has been rejected, and mucus, sometimes bile-stained or even tinged with blood, may be vomited. Diarrhœa also in such cases is more severe, and the constitutional disturbance is considerable. The infant quickly becomes hollow-eyed with sunken fontanelle, and lies apathetic and exhausted.

In these severer cases there is probably usually some inflammatory or catarrhal change in the mucous membrane of the stomach, and the term *acute gastric catarrh*, or even *gastritis*, might properly be applied to them.

Prognosis.—In young infants such an attack is always serious and may end fatally; but in most cases, if the cause is removed, the illness ends favourably. Infants who have had such an attack are very liable to relapse with the slightest error in feeding; and sometimes recovery is even less complete. The vomiting becomes less frequent, but does not disappear, the attack ceases to be acute, but becomes still more troublesome as a chronic condition.

Chronic vomiting is common, especially in hand-fed infants. In many cases, no doubt, it signifies a *chronic gastric catarrh*, but in many more it is due to a chronic dyspepsia, a functional disturbance, without organic changes, in the mucous membrane. Vomiting may persist for many weeks with no evidence whatever of organic lesion, and all one can say is that there is undue irritability of the stomach. As mentioned above, it seems likely that a morbid habit may be established which may account for the intractability of some cases.

To whatever cause the vomiting be due, except in the almost physiological repletion-vomiting of early infancy, the child soon begins to lose weight, its growth is arrested, it becomes pale and fretful. The appetite is sometimes large, sometimes diminished, and often there is pain and screaming after meals. The tongue may be furred, and sometimes the breath has a sour odour, and there are sour-smelling eructations. The bowels are often irregular, attacks of diarrhœa alternate with costiveness; but in many cases there is a chronic diarrhœa, and the condition is really a chronic gastro-intestinal catarrh (*vide* Chronic Diarrhœa, p. 103). Vomiting may occur two or three times a day, or after almost every meal. Unlike the repletion-vomiting which occurs immediately after, or even before the meal is finished, the vomiting of dyspepsia or catarrh usually follows after an interval of a quarter or half an hour, or more. In the worst cases the infant gradually becomes weaker and more emaciated; the skin is dry and hangs in folds; thrush appears in the mouth, and either from exhaustion or from broncho-pneumonia, or from some other complication, the child dies. In other cases the child slowly recovers, but remains delicate, and requires the utmost care in feeding for many months.

Morbid Anatomy.—As a rule the stomach shows nothing abnormal to the naked eye. It may be in a state of dilatation or contraction. In the worst cases, those of acute gastritis, the mucous membrane sometimes shows intense purplish congestion, usually over a limited area, sometimes also small hæmorrhages are seen in its substance, and very rarely small follicular erosions, while shreddy brownish material, probably altered blood or blood-stained mucus, is found adhering to it. The microscope shows shedding of superficial epithelium, small-cell infiltration both of the superficial and of the deeper parts, and considerable dilatation of vessels. In the more chronic cases, where no change whatever is detected by the naked eye, microscopic examination often reveals definite changes in the mucous membrane, especially when the viscid, mucus-like character of the vomit during life has suggested some gastric catarrh. The changes are similar to those found in the acute cases, except that they are less in degree, and the inflammatory infiltration shows more or less advanced organisation with the development of fibrous tissue which has partially replaced the glands of the mucosa. They are, in fact, exactly similar to those found in the intestine in some cases of diarrhœa (*vide* p. 107). The importance of these changes is in the light they throw on the intractable wasting which too often follows chronic vomiting in an infant.

Dilatation of the Stomach is present in some of these cases; perhaps especially where a chronic gastric catarrh is associated with rickets; the writer has found considerable dilatation in such cases. It has been shown that in the chronic dyspepsia of infants the food remains in the stomach longer than normal, and sometimes even clinically enlargement of the stomach can be detected, but anything like a chronic dilatation of the stomach, such as would give rise to the characteristic symptoms associated with such a condition in adults, is practically unknown in infancy, except in such obstructive conditions as congenital hypertrophy of the pylorus.

Treatment.—Vomiting, gastric in origin, is nearly always due to some fault in feeding, and sometimes all that is needed is a careful revision of diet. Where it is a symptom of diseases other than those of the gastro-intestinal tract, usually no special treatment is required. In an acute attack of vomiting due to faulty feeding, the evacuation of the irritant is usually sufficiently accomplished by the vomiting, but in some cases recovery may be hastened by making the evacuation more thorough, either by washing out the stomach through a soft catheter passed through the nose or mouth, or, as recommended by Dr. Holt, by giving the infant warm water in large quantity as an emetic. In severe cases of vomiting it is advisable to stop all feeding by the mouth for a few hours, and then to feed only with very minute quantities at short intervals. A teaspoonful of cold water with 5-10 drops of brandy every half-hour will be quite enough for a few hours; sometimes hot water seems more useful. Infants at the breast must only be allowed to suck for a very short time, and in some cases it is better to try albumin water or whey in small quantities for some hours before returning to the breast. Albumin water (the white of one raw egg cut up with scissors and shaken with half a pint of water, strained, and flavoured with a small quantity of cinnamon water or dill water) is often useful for a short time in any case of acute vomiting. Few foods are more valuable either in acute or chronic vomiting than whey, especially sherry-whey (made by heating half a pint of milk just to the boiling-point, then adding a good wineglassful of sherry, heating to boiling-point again, and when the curd has settled straining through muslin). A teaspoonful, gradually increased to a tablespoonful, of whey may be given frequently, and sometimes the addition of one or two drops of sal volatile to the plain whey checks the vomiting. When the attack has passed off the digestion is often much impaired, casein particularly seems to be digested with difficulty, and it may be necessary to peptonise the milk, or to use a humanised milk for a time.

In chronic vomiting the feeding is generally the most important consideration. Sometimes one food, sometimes another is successful, and it is often necessary to try several before a suitable one can be found. Whey, peptonised milk, humanised milk, prepared foods, especially the malted foods, are all useful at times. Whatever food is given, it must be given in small quantities, at short intervals, according to the age.

In any case of vomiting in which all food is persistently rejected, feeding through a tube passed down the œsophagus, either from the nose or mouth, is worthy of trial.

Sedative drugs are often of value, especially in acute cases after the stomach has been emptied. A mixture of bismuth and soda is generally useful, and a small dose of tinct. opii (♏ ¼-½ three times a day for an infant six months old) may be added to this, or hydrocyanic acid (♏ ¼ for an infant four months old) with sod. bicarb. and some aromatic may be given.

In the chronic cases these drugs are of less value; and small doses of gray powder given two or three times a day, alone or with soda, are more likely to be useful; sometimes papain (gr. j.-ij.) or pepsin (gr. j. with three or four meals a day) will do good by assisting digestion. Where the vomit is particularly sour or frothy, creasote is sometimes very effectual. Very severe and persistent vomiting, especially in acute cases, may sometimes be checked by counter-irritation in the shape of a mustard plaster to the epigastrium.

The value of stomach-washing is perhaps hardly sufficiently appreciated. Vomiting may be checked by only one or two washings with plain hot water or with a weak solution of sod. bicarb. (gr. ij. to ℥j.). This is specially useful where the presence of mucus in the vomit shows that there is some gastric catarrh; where there is evidence of fermentation, resorcin (1 in 1000) may be used for the washing.

Recurrent Vomiting (*Periodic or Cyclic Vomiting*).—It is convenient to mention this rare disorder here, although it is probably rather a nervous than a digestive disorder. It begins sometimes in infancy (in one case under the writer's notice it began at the age of six months), but more often after the first dentition. It is most common in children of nervous, excitable temperament.

Symptoms.—At intervals of a few months attacks of vomiting occur without apparent cause. The child is languid and depressed. Its face is sallow, the bowels are costive, and in some cases the motions are pale as if there were some deficiency of bile. Every attempt to take food is followed almost immediately by vomiting; retching is sometimes violent. The temperature may be normal throughout or somewhat raised, especially at the onset. After two or three days the vomiting usually ceases, and the child rapidly recovers, but sometimes the attacks last ten days or longer, and exhaustion may be so great as to threaten life. Such attacks may recur for several years; in the case mentioned above the attacks had recurred three or four times a year for five years.

It seems probable that these attacks are nervous in origin; certainly they seem to be little if at all influenced by such treatment as is useful in the vomiting of gastric catarrh. It has been suggested that they resemble migraine in their pathology, and Dr. Gee records a case in which the mother suffered from typical migraine.

Diagnosis.—The nature of a first attack can scarcely be recognised; the absence of any apparent cause and the intractability of the vomiting might suggest that it was of this character. A diagnosis can only be made with certainty after repeated attacks have shown the tendency to recurrence. To distinguish it from early meningitis may be extremely difficult, but headache is usually absent in recurrent vomiting, and is never likely to be so severe as in meningitis; later the absence of paralytic symptoms and of ophthalmoscopic changes will assist the diagnosis.

Treatment.—In the intervals all excitement and over-exertion, bodily and mental, especially school pressure, must be avoided. Healthy outdoor exercise short of fatigue should be encouraged. Diet should be simple. Intermittent courses of arsenic and bromides should be given.

During an attack the strength must be supported by careful feeding and stimulants if necessary. Fluids in very small quantities, iced or peptonised, may be tried, but in severe cases rectal feeding is necessary. The bowels should be opened freely by repeated small doses of calomel. Sedatives such as bromides, belladonna, or hydrocyanic acid, or counter-irritation to the epigastrium, may be tried, but usually the vomiting runs its course uninfluenced by drugs.

DIARRHŒA

(*Gastro-intestinal catarrh ; Gastroenteritis*)

Under this head will be described those disorders of the gastro-intestinal tract in infancy in which diarrhœa is the prominent symptom. In a large proportion of cases, however, the stomach is affected with the intestine, and vomiting may be quite as prominent a symptom as the diarrhœa. Various classifications of these disorders have been attempted, some based on clinical, some on pathological grounds, but the subject is one of great difficulty, for while it is evident that the cases fall into certain clinical groups, these groups are by no means well defined, and pathologically they tend to run one into the other. Diarrhœa may be acute or chronic. Acute diarrhœa may perhaps most usefully be divided into (1) Simple, (2) Inflammatory, (3) Choleraic.

Ætiology.—Before describing the various forms of diarrhœa it may be well to say something about their general ætiology; for our knowledge on this point is at present so vague that it would be useless to attempt to associate particular causes with particular forms except in a very general way.

There is no age at which diarrhœa is more prevalent or more fatal than that of infancy ; but the liability is much greater in the first year of life than in the second, the mortality also is much heavier in the first year. Statistics taken by the writer at the Hospital for Sick Children showed that 90 per cent of the fatal cases of infantile diarrhœa were under twelve months old. Girls and boys are about equally affected.

The influence of season is very marked. Scattered cases occur throughout the year, but during the hot summer months there is an enormous increase, both in the prevalence and in the mortality of infantile diarrhœa. The actual seasonal variation in any particular place or year varies with the meteorological conditions; in London the maximum prevalence would seem to be usually from the beginning of July to the middle of October.

There is manifestly a very close relation between a high mean temperature of the atmosphere and infantile diarrhœa, but what this relation may be it is difficult to ascertain exactly. Probably the high atmospheric temperature favours the growth of certain bacteria which are capable of exciting diarrhœa ; these reach the intestine in the food, particularly in cow's milk which has not been properly prepared. But what the source of these bacteria may be is unknown ; it has been suggested that they are from the soil, and it has been noticed that the summer increase of diarrhœal mortality follows the curve of soil-temperature as shown by the four-foot earth-thermometer (Ballard).

At any time of the year infants who are entirely or even partially hand-fed are much more liable to diarrhœa than those who are entirely breast-fed ; and this difference is no doubt due chiefly to the almost complete sterility of human milk as drawn by the infant from the breast, while fresh cow's milk as supplied commercially almost invariably contains some bacteria.

But other factors are also present in the diarrhœa of hand-fed infants ; the food is often, especially amongst the poorer and more ignorant classes, of a most indigestible kind; and in all classes it is apt to be given in too large quantity and at too short intervals.

The mortality from infantile diarrhœa is greater in town than in country districts, but it would seem that this difference does not depend entirely on density of population; poverty and defective sanitation are potent causes.

There can be little doubt that infantile diarrhœa is sometimes contagious; cases have been observed where it has seemed to spread from infant to infant, and also to nurses, but the possibility of a common source of infection, especially in the milk-supply, must be carefully excluded.

Of recent years attempts have been made to isolate from the very numerous bacteria which are always present in the intestine a specific micro-organism as the cause of the diarrhœa, especially the summer diarrhœa of infants, but hitherto the results have been quite inconclusive.

Dr. Klein has recently isolated a micro-organism, the bacillus enteritidis sporogenes, which there is reason to believe plays an important part in the causation of some of the acute cases of summer diarrhœa; but other micro-organisms have also been found with more or less constancy, and it seems probable that there may be several different micro-organisms which are all equally capable of exciting a gastro-intestinal catarrh.

The manner in which these bacteria cause diarrhœa is also probably not the same in all cases: in some they produce chemical changes in the food either outside the body or in the intestine, so that the altered food is an irritant; in others they themselves produce toxins which not only act as an irritant to the mucous membrane, but also when absorbed may cause some of those profound disturbances, especially of the nervous system, which are so marked in some cases of diarrhœa.

But while it is almost certain that a very large proportion of the cases of diarrhœa in infancy are due to micro-organisms, and are therefore "infective,"—and it must be understood that the mildest summer diarrhœa and the most virulent choleraic diarrhœa are in this sense equally "infective," though probably due to different micro-organisms,—one recognises clinically many cases in which there is no reason to suppose any such cause at work.

Indigestible food in the intestine may act as a mechanical irritant. Certain chemical poisons are sometimes the cause of diarrhœa, and apart from the ordinary aperient medicines some drugs given medicinally for other purposes, *e.g.* ammonium carbonate or arsenic, may act thus.

Most of the specific fevers are occasionally associated with diarrhœa; thus whooping-cough, measles, scarlet fever, and diphtheria are all sometimes complicated by severe diarrhœa. Typhoid and tuberculous ulceration of the intestine will not be considered here, as they fall into a somewhat different category, but in any given case of diarrhœa they must, of course, be considered as possible causes.

Diarrhœa is very common with broncho-pneumonia in infancy; and it is often difficult to be sure which was the primary disease; here, as in the specific diseases just mentioned, the diarrhœa may be "infective" in character, and perhaps the same explanation applies to the diarrhœa which often complicates suppurative conditions, such as pyæmia or empyema.

The part played by dentition in the causation of diarrhœa is obscure, but it is certain that the eruption of a fresh tooth is frequently associated with diarrhœa; possibly the nervous disturbance which so often accompanies dentition may, like fright or excitement in older children, affect the intestine.

Lastly, the effect of chill must be mentioned; chronic diarrhœa especially sometimes seems to be prolonged, if not actually caused, by exposure to cold, and in infancy this cause is too often present owing to the insufficient covering for arms, legs, and abdomen which custom demands.

SIMPLE DIARRHŒA (*Intestinal Indigestion, Gastro-intestinal Catarrh*).—In its mildest form simple diarrhœa is quite a transient and slight affection. The infant, who has previously been quite well, seems restless, and perhaps shows signs of colicky pain, screaming and drawing up its legs. The bowels are open five or six times in the day; the fæces at first are only slightly looser than normal, and probably contain undigested casein or other food-stuff, but soon they become more liquid, and perhaps green and offensive with some mucus. Frequently some vomiting precedes the diarrhœa, showing that there is some gastric as well as intestinal irritation. The temperature may be raised to 100° or 101° at first, but quickly becomes normal, and often is normal throughout. Even with a very mild attack the infant quickly shows that it is ill; it ceases to be playful, and if it has already begun to crawl or walk, it may give up these accomplishments altogether; as the mother says, "it has gone off its legs," it only wants to lie quietly in its cot or in its mother's arms. At the same time it is pale and dark under the eyes; its tongue is slightly furred, and it may refuse its food, or drink greedily as if it were thirsty.

But the condition rapidly improves; sometimes after only a few hours, sometimes after two or three days, the stools become less watery, and gradually resume their normal colour and consistency. The infant remains pale and fretful for a day or two after the diarrhœa has ceased, but speedily regains its usual health.

Such is a mild attack of diarrhœa in an infant; and where the illness is due, as commonly happens, to some irritating food-substance in the intestine, the evacuation of the irritant usually brings the attack to an end. Too often, however, these mild symptoms do not pass off as soon as one expects, the diarrhœa continues day after day, and what began as a simple diarrhœa becomes the more serious condition which is called inflammatory diarrhœa.

INFLAMMATORY DIARRHŒA (*Febrile Diarrhœa, Gastroenteritis, Ileocolitis*).—Such a heading necessarily includes several conditions which are more or less distinct pathologically, but as these cannot with any accuracy be distinguished clinically, it seems more useful to describe them together. Many cases of "summer diarrhœa" are of this variety.

Often the condition begins like a simple diarrhœa, but instead of subsiding in a few days the diarrhœa continues; the stools are liquid, greenish, or dark brown and offensive, and often show some mucus and streaks of blood. The bowels are open many times a day, and the buttocks and perinæum are soon red and raw with the irritation of the fæces. It is remarkable how quickly nutrition suffers in these cases; after even one day of diarrhœa, if it be at all severe, the muscles feel less firm, and the face looks less full, and in two or three days there is distinct loss of flesh, the child is pale and hollow-eyed, and the fontanelle depressed. The tongue is often quite clean, in fact, almost abnormally clean and red, or slightly furred with very red papillæ projecting through the white fur.

The temperature may be raised at the beginning of the illness, but afterwards in the less severe cases it is normal; if the diarrhœa be prolonged, and especially if there be much vomiting, it may be subnormal.

After two or three weeks of such symptoms the diarrhœa may gradually cease, and the child slowly mend; but at any time in such an attack, and sometimes, indeed, from the beginning, more severe symptoms may occur. The temperature rises and there is irregular pyrexia; the infant looks acutely ill, it is drowsy and apathetic, the cheeks are flushed, the tongue is furred. The abdomen is often full, and sometimes distinctly tender on palpation, especially along the course of the colon. The stools are very frequent, perhaps ten or twelve a day, and mucus stained a reddish brown or streaked with bright red blood is commonly present, pointing to affection especially of the colon. Vomiting is frequently associated with the diarrhœa, even where the lower part of the intestine seems to be chiefly affected.

Dr. Eustace Smith attaches considerable importance to loss of elasticity in the skin in these cases as evidence of deficient renal excretion, and undoubtedly the urine is diminished, as might be expected where food is so rapidly hurried through the intestine and so little absorption takes place. It is, however, equally certain that any actual nephritis is an exceedingly rare complication, in spite of the fact that a small amount of albumin in the urine is very common in all forms of diarrhœa in infancy.

Œdema not infrequently occurs, especially in the extremities, after the diarrhœa has lasted a week or more; this again must not be taken as necessarily indicating any renal complication; in some cases where it is marked the urine is perfectly normal, and even if albumin be present it is usually very slight; it seems probable that the œdema is rather due to a flagging circulation, combined with some hydræmic condition of the blood, and possibly with some degenerative change in the vessels.

Eventually exhaustion becomes extreme; the infant lies quite apathetic, almost in a stupor, with sunken eyes and depressed fontanelle; the eyelids are only half-closed in sleep, and a film of muco-pus collects on the cornea, which may become dry and ulcerated. The extremities are cold, the pulse is feeble and very rapid. The temperature, as the infant lies in this state of collapse, may be subnormal, but often when taken in the rectum it is found to be high; there may even be hyperpyrexia in spite of the coldness of surface. Death results usually from exhaustion, but in some cases is preceded by convulsions, or by symptoms like those of meningitis.

These cases, however, are by no means always fatal; the diarrhœa may gradually cease and pyrexia disappear, and the infant may gradually regain its former nutrition. Too often, however, recovery is less complete; sometimes, although the acute diarrhœa has ceased, the bowels continue to be open three or four times a day, or are irregular, constipation alternating with looseness; the fæces show undigested curd, and are perhaps greenish and offensive with some mucus; the child gradually becomes puny and wizen-faced, and after weeks or months of slow emaciation dies of exhaustion. Sometimes, although the bowels become regular, the infant steadily wastes, and no manner of feeding seems to influence the wasting which eventually proves fatal. In other cases, after the acute diarrhœa has been checked, the child seems to have made a good recovery, but for months, and sometimes even for years afterwards, the slightest fault in diet causes some diarrhœa, or the appearance of mucus in the stools with more or less disturbance of general health.

CHOLERAIC DIARRHŒA (*Cholera Infantum, Septic Diarrhœa*).—This form of diarrhœa is more especially limited to the summer season than are the preceding forms. The name is usually given to any case of summer diarrhœa in which the stools are watery, and the illness runs a very acute course, lasting only a few days or even a few hours. It has been suggested that the name should be reserved for those cases in which the stools have a rice-water appearance, but as this symptom does not, so far as our present knowledge goes, indicate any special bacteriological difference, and may or may not be present in cases which otherwise run an exactly similar course, there seems to be no reason for adopting this limitation. It will, of course, be understood that there is no connection between so-called "cholera infantum" and Asiatic cholera.

Symptoms.—These, as already mentioned, are characterised by their extreme severity and acuteness. In the worst cases, an infant, often well nourished and apparently in perfect health, is suddenly seized with diarrhœa. The stools, at first semi-liquid and yellowish, rapidly become thin and watery, sometimes brownish and very offensive, usually almost colourless, like rice-water, with very little smell. Vomiting is severe, all food is rejected almost directly it reaches the stomach. In a few hours the alteration in the face is striking, the plump pink cheeks have become pale and flabby, the eyes are deeply sunken, and the brows often knitted as if with pain. The fontanelle is deeply depressed, the tongue is dry and brownish red, and the infant is continually licking its lips in its distressful thirst. The abdomen is deeply hollowed out, and the skin over it lies in loose, wrinkled folds. The child flings its arms restlessly about, and rolls its head from side to side, or lies in an apathetic condition, taking no notice of its surroundings, and gradually sinking into actual stupor. Respiration is often sighing; the temperature is usually high, but may be subnormal. The urine is diminished, and may even be suppressed for many hours, and often shows more or less albumin.

After two or three days, or, in the worst cases, after only a few hours, exhaustion is extreme, the extremities become cold and blue, the face, and especially the nose, has a pinched look and gray colour, the pulse becomes very rapid, 180-200, and barely perceptible at the wrist, and the child dies.

The course of the disease varies somewhat in different cases. The onset may be less acute than that described above; the infant is ailing for a few days with what appears to be a simple diarrhœa, and no alarm is felt until a sudden increase in the severity of the diarrhœa, with the appearance of watery colourless stools and the onset of uncontrollable vomiting, shows how serious the illness is. In other cases the choleraic symptoms supervene in the course of a chronic diarrhœa or intestinal indigestion which has lasted for many weeks or months, the temperature which has hitherto been normal suddenly rises, diarrhœa and vomiting are profuse, and death occurs in a few days (see also "Cholera Nostras," vol. ii.).

CHRONIC DIARRHŒA is in many cases the sequel of acute diarrhœa, but it may occur independently of any acute attack, and may be quite insidious in its onset. In these insidious cases the diarrhœa is perhaps most often the result of faulty feeding; it is, in fact, the manifestation of a chronic dyspepsia. In some there is also some constitutional defect present, sometimes syphilis, but much more often rickets, which is so powerful a predisposing cause of catarrh of mucous membranes. Chronic diarrhœa is not uncommon as a sequela of whooping-cough and of measles. In the children of the poor, exposure to cold and generally faulty hygiene play an important part in its production. Under this head come many of the cases of so-called "consumptive bowels," a vague, inaccurate term which it is to be hoped will die out ere long; very few of the cases to which it is applied show any tuberculous lesion, and many of those who use the term simply mean by it a disorder of the bowels associated with wasting. Chronic diarrhœa may be the result of tuberculous ulceration of the intestine, but this is quite the exception in infancy.

Symptoms.—The diarrhœa may have persisted since an acute attack, probably as a much slighter looseness of the bowels, which are open four or five times a day, with some undigested food in the fæces. But often no definite date can be assigned for its onset, and so insidious may it be, that the mother has only noticed the wasting, the condition of the bowels has not attracted her attention. On inquiry, it is found that the bowels are open too frequently, and the stools are unhealthy. The character of the stools varies considerably. In some cases of chronic diarrhœa the stools are semi-liquid, deep brown, and very offensive; in others they are pale yellowish or drab coloured, and pultaceous; in others again there are small lumps of white curd mixed with very small pieces of dark green fæcal material very like chopped spinach. The green colour is probably due to the presence of biliverdin; alkaline decomposition in the intestine changes bilirubin to biliverdin (Pfeiffer); the fæces may be acid when passed, but the acidity is not sufficient to change the colour. The suggestion that a specific micro-organism causes the colour (Lesage) lacks evidence.

The presence of mucus, especially if stained with blood, points to affection of the colon, and if the blood occur in bright red streaks, it is probable that the lower portion is specially affected. Those cases in which the colon is much involved are further distinguished by the presence of tenesmus, sometimes by some tenderness over the large intestine, and also by a tendency to prolapse of the rectum, which is, however, more marked in children two to four years of age than in actual infancy.

In all cases the general health suffers considerably. The infant, who was good-tempered and playful before, becomes fretful, miserable, and apathetic. If he be at the age of learning to talk, he ceases to make progress, and may lose entirely the few words he has acquired, and a child who has just learnt to walk with the aid of a chair may even be unable to stand. Nutrition is impaired very quickly. The infant rapidly wastes, the skin is dry and inelastic, and can be pinched up in loose folds. After a few weeks of chronic diarrhœa the emaciation is often extreme, the face is shrunken, and looks aged and wrinkled, there is a deep furrow passing downwards from just above the ala nasi on each side, and forming a crescent round the angle of the mouth, so that the lips and mouth seem to be prominent, and the general appearance is not unlike that of a monkey. The skin, both of the face and trunk, is sometimes of a muddy brownish colour, sometimes very white, the edges of the eyelids look red and sore, and there is frequently some eczema behind the ears. The fontanelle is depressed, the cry is a feeble whine, the extremities are often cold and blue. The tongue is quite clean in many cases, but may be slightly furred. The abdomen is usually retracted, and through its wasted wall the coils of intestine can be seen; slight peristalsis may also be visible. Sometimes the abdomen is full from the presence of gas in the intestine, and at the end of the illness an acute abdominal distension may occur and hasten the fatal result. Flatulent eructations are often present, and sometimes have an extremely disagreeable sour odour. As in other forms of diarrhœa, especially when there is any neglect in changing the diapers and in keeping the parts clean and dry, the buttocks and perineum are apt to become red and excoriated.

In this condition, perhaps, more commonly than in acute diarrhœa, œdema first of the extremities and then of the whole body is likely to occur, and is usually a symptom of the gravest significance; purpura also in spots or larger patches is very common towards the end of the illness, beginning often on the abdomen, or on the upper part of the chest or neck. After these symptoms have appeared the end is not usually long delayed.

Sooner or later the diarrhœa becomes more severe, the bowels are open ten or eleven times a day, and the evacuations are more liquid; vomiting, which in many cases has only been occasional and slight, or may have been absent throughout the illness, now becomes frequent, and the temperature which has hitherto been normal may rise, or may drop to subnormal. In some cases there is irregular pyrexia for several days before death; in others the temperature remains normal until a few hours before the fatal issue, when it rises to 107° or 108°. With the onset of these symptoms the child falls into the state of collapse, which has already been described, and death results as a rule from exhaustion.

But although chronic diarrhœa in an infant is always a serious condition, and always calls for a very guarded prognosis, it is not always fatal. With careful treatment the bowel may be coaxed back to a healthier action, and the child slowly recover, but in such cases, even more than in those which recover from inflammatory diarrhœa, the child is likely to be delicate for a long time after the attack, and relapses are extremely common.

Complications.—In many cases of diarrhœa, whether acute or chronic, but especially in the more severe cases of acute inflammatory diarrhœa, bronchitis and broncho-pneumonia are very liable to occur. The association is always a dangerous one, and in some cases the fatal result appears to be directly due to the broncho-pneumonia. In chronic diarrhœa the infant is often so feeble and the broncho-pneumonia comes on so insidiously, that it produces but little alteration in the symptoms, and may only be discovered post-mortem.

Collapse of the lung, although very commonly found after death in fatal cases of diarrhœa, can seldom be detected clinically.

Thrush is frequently associated with diarrhœa, especially in the more chronic cases; it is of less serious import than mothers usually imagine, but where the soreness of the mouth interferes with the taking of nourishment it may add to the seriousness of the condition.

Otitis media is liable to occur with diarrhœa as with any other severe illness in childhood, no doubt owing to the prostration favouring the accumulation of micro-organisms in the pharynx and their extension up the Eustachian tubes. The complication is worth bearing in mind, as it may simulate cerebral symptoms and cause much distress, which is easily relieved by hot applications, or, if necessary, by puncture of the membrana tympani.

Nervous complications are not rare in severe diarrhœa. The commonest are convulsions. These sometimes occur at the onset of the disease, but much more often during the last few days of life. In many cases the child falls into a sort of continuous convulsive state, rather than an actual convulsion; it lies apparently unconscious, with its eyes turned upwards or to one side, its hands and legs twitching slightly, and an occasional twitch of the facial muscles, and in this condition it may remain for some hours before death occurs. The occurrence of convulsions in the course of a severe diarrhœa is always a very serious symptom.

Sometimes, both in severe cases of inflammatory diarrhœa and in choleraic diarrhœa, where collapse and exhaustion are extreme, the child falls into a condition which resembles in some respects meningitis. It has been called "spurious hydrocephalus." The child becomes almost completely unconscious, the head may be retracted and the limbs rigid, there may be some strabismus, and even the sudden scream of meningitis may occur, the respiration is irregular, the pulse feeble and rapid; but with all these symptoms the fontanelle is depressed. As a rule such cases are speedily fatal. No naked-eye lesion is found in the brain to account for the symptoms, which it seems probable are due to some toxæmia from the intestinal infection.

Thrombosis of cerebral sinuses is a very rare complication. It occurs usually in cases where there is great exhaustion; it is but seldom recognised during life, the symptoms are very variable; paralysis of cranial nerves, weakness of limbs, and convulsions have been observed, but sometimes there are no symptoms at all.

An exceedingly rare complication of diarrhœa is nephritis. It occasionally occurs in very severe cases of cholera infantum; otherwise, except for some slight cloudy swelling in cases where there has been much pyrexia, the kidney is almost invariably healthy, although, as already pointed out, slight albuminuria is common. It is possible that in some cases this albuminuria is the result of mechanical irritation of the pelvis of the kidney by uric acid sand, which is very frequently found in the urine during life and in the pelvis of the kidney after death in these cases.

Prognosis.—In all cases of diarrhœa in infancy the prognosis must be guarded. The case which at its onset looks like a mild case of simple diarrhœa may in a few days merge into the severest form of inflammatory diarrhœa. At the same time it must be remembered that a simple diarrhœa may be very severe in its onset, and may produce considerable exhaustion, with sunken eyes and depressed fontanelle, especially in very young infants, but after a few hours the symptoms may gradually subside, and in a few days there may be complete recovery. In all forms of diarrhœa, the younger the infant the less favourable is the prognosis. Continued high temperature, whether regular or irregular, in any case of diarrhœa is a serious symptom; it usually means that the case is one of inflammatory diarrhœa, and sometimes points to the grosser lesions, such as ulceration and membranous inflammation, which are found in some cases of inflammatory diarrhœa. Tenderness of the abdomen also points to an inflammatory condition, and when it is well marked is an unfavourable symptom.

Profound nervous disturbance, especially apathy or stupor, is a very grave symptom. Convulsions are serious, but do not necessarily mean a fatal result; their significance is less grave when they occur at the onset of the diarrhœa than when they occur in the stage of exhaustion and prostration.

Choleraic diarrhœa is always a grave disease; a very large proportion of such cases are fatal.

Diagnosis.—In some cases of chronic diarrhœa it is extremely difficult to determine whether the diarrhœa is due to tuberculous ulceration of the bowel. A gastro-intestinal catarrh from faulty diet is very much commoner in infancy than tuberculous disease of the intestine. In infants under six months of age tuberculosis is decidedly rare, and even up to the age of nine months it is not common. The association of irregular pyrexia with a chronic diarrhœa suggests a tuberculous lesion, especially where there is a strong family history of tuberculosis, but the strongest evidence is palpable enlargement of the mesenteric glands or the presence of some definite tuberculous lesion elsewhere. In most cases the question cannot be decided during life, and one can only say that the vast majority of the cases of chronic diarrhœa in infancy are non-tuberculous. Typhoid fever is so rare in infancy, and when it does occur is so much more often associated with constipation than with diarrhœa, that it is hardly likely to cause any trouble in diagnosis; the continuous or remittent pyrexia lasting ten days or a fortnight will usually serve to distinguish it from chronic gastro-intestinal catarrh, and other symptoms of typhoid, enlarged spleen, rose spots, or a positive result with Widal's reaction, may assist the diagnosis.

The frequency with which mucus and streaks of blood are present in the stools in diarrhœa with affection specially of the colon is noteworthy; these appearances do not always indicate an intussusception, but on the other hand the occurrence of an intussusception has been mistaken for the onset of a gastro-intestinal catarrh, and the presence of mucus and blood on the stools has been made light of, with disastrous result. Such an error can be avoided only by careful examination of the abdomen for the presence of the characteristic tumour.

The presence of ulceration in the bowel cannot be determined clinically; it is not even possible to determine with any accuracy during life which part of the intestine is affected. Such terms as ileocolitis, ulcerative or membranous colitis, have a definite meaning for the pathologist, but are of very little practical value for the clinician. In a general way it may be said that an acute course with a very high temperature, the passage of mucus and streaks of blood in the fæces, and tenderness along the course of the colon, point to some intense inflammation of the large intestine, but a case may show all these symptoms during life, and yet show nothing post-mortem beyond slight congestion of the mucous membrane of the intestine.

Morbid Anatomy.—The most striking feature in the morbid anatomy of the diarrhœa of infancy is the extreme disproportion between the naked-eye lesions found post-mortem and the severity of the clinical symptoms. In the most acute form of all, choleraic diarrhœa, the intestine usually shows nothing abnormal beyond pallor of the mucous membrane. In many cases of inflammatory diarrhœa there is no naked-eye lesion whatever; in many there is only slight congestion of the mucous membrane in parts of the ileum and colon. A few cases show some undue prominence of the solitary follicles in the large intestine, so that its mucous membrane appears to be studded thickly with little round bosses, and in such cases there is sometimes also swelling of the Peyer's patches in the lower part of the ileum. Ulceration is but rarely present (the writer found it in 5 per cent). In some cases small round ulcers are present in the ileum; they are mostly shallow, with no thickening of their edges, and are usually quite independent of the Peyer's patches; sometimes only four or five are present, sometimes a score or more. In other cases a much more extensive irregular ulceration occurs in the colon, and more rarely in the lower part of the ileum; the ulcers are usually superficial, with no thickening at their edges, but the surrounding mucous membrane may be intensely congested and swollen. Such ulceration is often limited to one part of the colon, it may be at the cæcum and on the lips of the ileo-cæcal valve, or in the lower part of the colon, or even in the rectum. A still more rare condition is membranous inflammation of the colon, in which irregular areas of superficial ulceration are covered by a grayish white membrane, very like that seen on the fauces in diphtheria.

In some of the more chronic cases of diarrhœa a curious appearance like that of the chin after shaving is seen, areas of mucous membrane in the colon or in the lower part of the ileum are closely stippled with black dots, giving the "shaven beard" appearance, probably due to the deposit of pigment perhaps from minute blood extravasations in the small glands or in the superficial part of the mucous membrane.

Microscopic examination of the intestine may show well-marked lesions even where there is nothing abnormal to the naked eye. The earliest change is a round-cell infiltration of the mucosa, with some shedding of the epithelium; in more advanced cases the sub-mucosa is also infiltrated, the vessels are distended, and there is distinct increase in size of the solitary follicles and of the Peyer's patches from similar round-cell infiltration. Sometimes the mucosa is found at some parts to have disappeared over a collection of small round cells which form the base of an ulcer. In the most severe cases there may be considerable exudation of fibrin together with the round-cell infiltration, and the presence of such fibrin on the surface of the mucosa or over an ulcerated area produces the membrane which is seen in some of these severe cases.

In chronic diarrhœa similar changes occur, but repair has already taken place to some extent, the inflammatory infiltration has become organised, and a newly-formed fibrous tissue is seen separating and compressing the glands of the mucous membrane, which have in this way disappeared. An actual cirrhosis of the mucous membrane thus takes place, and the affected areas are no longer able to absorb properly. These histological changes account for the intractable wasting which may follow a chronic diarrhœa.

Treatment.—(*a*) Prophylactic.—The first and most important of all measures is the continuance of breast-feeding, and breast alone, until the age of at least nine months, and it is wise to prolong it for two or three months longer if in this way weaning during the hot summer months, especially July and August, can be avoided.

In hand-fed infants strict care in diet (*vide* "Infant Feeding"), and in particular the sterilisation or pasteurisation of the milk, will do much to avert the risk of diarrhœa. The use of the old-fashioned feeding-bottle, with its long rubber tube coated inside with stale milk and bacteria, is to be strictly forbidden, and the modern boat-shaped bottle substituted.

There can be no doubt that attention to the sanitary arrangements of the house, and in particular of the nursery, is of the greatest importance. Some of the worst cases of diarrhœa in infants and in young children occur where sanitation is in some way defective, it may be from some leakage of a soil pipe, or from defective traps in a water-closet outside their room. Ventilation must be free; soiled diapers must never be allowed to remain in the nursery. Town children may with advantage be sent away to the country or seaside during the hot season; the cooler and purer air of the country certainly has a considerable value as a curative agent when diarrhœa is already present, and probably is also of great value in prophylaxis.

The influence of chill in the production of diarrhœa in infants must not be forgotten. Knitted drawers worn over the diaper should cover the abdomen and the upper part of the thighs, and when the child goes out woollen gaiters reaching well up the thighs, or better still woollen drawers reaching down to the ankles, should be worn, not only on winter days, but also on the chilly days of autumn and spring.

(*b*) Therapeutic.—In the treatment of diarrhœa in infancy diet is often of greater importance than drugs. In a large number of cases, especially of chronic diarrhœa, the feeding is found to be entirely unsuitable, and the first step, sometimes indeed the only one necessary, is to correct the diet in accordance with the age of the infant. It will often happen, however, that some special diet is required as a temporary measure. In most cases of acute diarrhœa milk must be omitted altogether for some days; but where it is thought advisable to continue it, the milk should be well diluted, and lime water should be freely given with it, for, apart from its value in increasing the digestibility of the milk, lime water itself certainly has some controlling influence on the diarrhœa. Rice water may be used as a diluent; barley water seems in some cases, especially perhaps in the younger infants, to keep up the diarrhœa, sometimes, however, it agrees well, and may be used alone for a day or two where milk cannot be given.

Broths are very useful, particularly veal broth and chicken broth; mutton broth can be used where these cannot be obtained. Fat must be removed from the meat before the broth is made, and any fat in the broth must be strained off. Beef tea is better avoided; it is apt to make the diarrhœa worse and the stools offensive. Raw meat juice is sometimes useful, but rather as an alternative with some other food, such as whey or barley water, than alone. Whey is particularly valuable where there is much vomiting (*vide* p. 97). In infants beyond the age of one year, and particularly in the more chronic cases of diarrhœa, thin arrowroot is valuable, and seems to aid in checking the disorder.

In very acute cases of diarrhœa, especially where there is much vomiting, and in choleraic diarrhœa, the drain of fluid from the child is very rapid and severe, and appears in many cases to be in part, at least, the actual cause of death. The immediate supply of fluid in such cases is a vital necessity, and the quantity is of much more importance than the quality. If fluid with some nutritive value, such as barley water or albumin water, can be given by mouth and retained, so much the better, but if not, plain sterilised water is almost as useful; and if even water cannot be retained when given by the mouth, or cannot be taken in sufficient quantities, it must be given either by rectum, a method not always available owing to the diarrhœa, or subcutaneously by several injections in various parts of the body. Intravenous injection is not an easy procedure in infants, and as the injections may have to be repeated several times it is simpler and better to inject the water hypodermically. Albumin water has been used for hypodermic injection, but this is unnecessary and involves some risk of inflammation. It is unnecessary even to use a saline solution, simple boiled water seems to serve equally well; as much as an ounce and a half or two ounces can be injected altogether, and the injection repeated in a few hours' time if necessary. Intraperitoneal injection of saline solution cannot be recommended; even with the most scrupulous care it is not free from serious risks, and although it has caused decided improvement within the writer's experience, it has little if any advantage over the much safer method of hypodermic injection.

Whatever method of feeding be adopted, the presence of vomiting in severe cases usually makes it necessary to give the food in small quantities at short intervals; and all food given by the mouth should be either cold or only just warm.

In almost all cases of acute diarrhœa, where there is much exhaustion, stimulants are necessary. In severe cases where there is collapse nothing is more effectual than a hot mustard bath. But unless this is given skilfully it may do more harm than good. Too often one has seen infants held with their backs under water, while the whole of the front of the chest and parts of the limbs are projecting wet above the surface of the water, simply getting chilled by evaporation. The water must be deep enough to cover the child completely up to its neck; one brimming tablespoonful of mustard (or more in proportion to the amount of water) should be mixed into thin cream with tepid water and then stirred into a gallon of water at a temperature of 100°; the infant should not be kept in the bath more than three minutes, and should then be taken out and immediately wrapped in a hot blanket, while it is rapidly dried with a hot towel without any exposure. It must then be put back to bed in a hot blanket with hot bottles.

Sometimes it is more convenient to use a mustard pack, the infant being wrapped for about ten minutes in a sheet wrung out of mustard and water in the proportion of 1-2 tablespoonfuls of mustard to the gallon.

Brandy or whisky should be given internally; in severe cases 10-15 drops may be given in a teaspoonful of water as often as every two hours to an infant three months old, and for an infant a year old as much as an ounce in the twenty-four hours may be necessary. A drop of sal volatile given occasionally in a teaspoonful of the food not only acts as a stimulant, but sometimes checks vomiting; it must not, however, be used too freely, as it may aggravate the diarrhœa.

An extremely useful stimulant where exhaustion threatens to prove fatal is strychnine administered hypodermically; ¼ minim of the liquor may be used for an infant three months old, and may be repeated after three or four hours. As a last resource, last because painful, brandy or ether (♏ x.-xx.) may be injected subcutaneously.

In simple diarrhœa a single aperient dose of castor oil or of calomel may be required to clear out the offending substance, and this alone may be sufficient; but usually medical advice is not sought until the bowels have already been open so freely that the irritant has probably already been removed, and sedatives are needed to allay the resulting catarrh. For this purpose opium is the most generally useful drug; the tincture may be given in doses of ♏ ⅙-¼ three times a day to an infant four months old, ♏ ½ three times a day at six months, and ♏ j. every four hours at twelve months. Bismuth is often useful with opium or alone, but it must be given freely; five grains is a suitable dose for an infant a year old; it is perhaps best given in suspension, with sodium bicarbonate. Astringents are sometimes useful in such cases after the acute symptoms have passed off. Some such mixture as the following may be tried:—Tr. catechu ♏ x., Spirit. chloroformi ♏ ij., Mist. cretæ ad ʒj.; or a mixture containing hæmatoxylum, or some preparation of tannic acid, may be given. But the results of such astringents in any form of infantile diarrhœa are disappointing; in many cases they do not seem to have the least effect. A combination of acid. sulph. dil. or acid. nitric. dil. (♏ ij.-iv.) with opium often does more good.

If the diarrhœa does not cease after a few days the following mixture may be found effectual:—Ol. ricini ♏ v., Mucilag. acaciæ ♏ xv., Aq. menth. pip. ad ʒj. There are few drugs which are more generally useful in the treatment of subacute or chronic diarrhœa than castor oil given in this way, especially if a small dose of opium, or, where the stools are offensive, a drop of liquor hydrargyri perchloridi, be added to it.

In many of the more severe cases of inflammatory diarrhœa, especially where the stools are green and offensive, gray powder is of great value, and often does much good in combination with Dover's powder; thus to an infant of six months a quarter of a grain of each may be given three times a day.

As to the value of antiseptics other than mercury there is some difference of opinion. In the most acute cases of summer diarrhœa the writer has not found them of much use; but in the less acute cases, and still more when the diarrhœa has become chronic, they are sometimes helpful. Perhaps the most useful of these drugs are the salicylates: salicylate of soda (gr. ij.-iv.) especially in combination with bismuth, or bismuth salicylate itself, has certainly done good in many cases, and these preparations have seemed on the whole more effectual than salol (gr. j.-iij.). Naphthalin (gr. j.-iij.), β-naphthol (gr. j.-iij.), glycerin acidi carbolici (♏ j.-ij.), and resorcin (gr. ij.-iv.) are all worthy of trial, and have been praised by various observers. The last mentioned is the most convenient, for not only is it readily soluble in water, but it is less disagreeable in taste than the others.

Creasote is specially valuable where there is much tendency to abdominal distension, a condition which often goes with frothy liquid stools; it may be given in half-minim doses every three or four hours with the castor oil mixture mentioned above. In cases where from the passage of mucus and blood, or from tenderness along the course of the colon, there is reason to believe that the colon is chiefly affected, irrigation of the colon may be of use. This may be done once or twice a day with plain warm water or with a solution of boracic acid, or the addition of tannic acid (gr. xx.-xxx. to the pint) may be tried. It is useful after the irrigation to give a small enema of starch with tincture of opium (♏ j.-iij.), and such an enema should be given in any case where great frequency of stools with much straining or prolapse of the rectum indicates excessive irritability of the lower part of the intestine. Occasionally irrigation seems to be of value in cases where there is no evidence of special affection of the colon; in acute cases of diarrhœa it has at any rate the recommendation that a certain amount of the fluid is likely to be absorbed.

In choleraic cases the vomiting is generally as serious as the diarrhœa, and often it is almost useless to administer drugs by the mouth. Good results have been obtained by washing out the stomach in such cases; and a mustard poultice to the epigastrium may help to check the vomiting. If opium can be retained it is well to give a dose of opium with brandy at first, and then bismuth may be tried. Frequent minute doses of gray powder have seemed to do good. Dr. Eustace Smith recommends a sixth of a grain of calomel every half-hour. In infants of twelve months or more hypodermic injections of morphia (gr. $\frac{1}{40}$ for a child of twelve months) have done good. Usually, however, these cases are so acute, and prostration is so rapid, that the most important part of treatment is free stimulation by one or other of the methods described above.

Chronic diarrhœa generally calls for dietetic treatment chiefly; but some drugs are extremely useful in this condition, and none more so than the castor oil mixture mentioned above. Antiseptics also, as already pointed out, are of more service in chronic than acute conditions. The combination of gray powder with Dover's powder is specially useful, and may be given with advantage in addition to the castor oil mixture. The astringents are generally useless after a diarrhœa has already continued many weeks.

LIENTERIC DIARRHŒA.—It is convenient to mention this disorder here although it occurs rather in children four to ten years of age than in infants. It differs from the forms of diarrhœa described above, inasmuch as it is quite independent of errors of diet, of infection, and of seasons. It is probably closely allied to the diarrhœa which occurs sometimes both in children and in adults as the result of fright or emotion. In a considerable proportion of these cases there is a family history of rheumatism, and the occurrence of lienteric diarrhœa in such cases is a manifestation of the nervous temperament which is so often present in the children of rheumatic families.

The characteristic symptom of this condition is the evacuation of the bowels immediately after every meal; sometimes, indeed, the desire to defecate comes on before the meal is finished. The motions are not necessarily loose, but often contain undigested food; as the mother says, "directly the child eats anything it goes through him." Sometimes there is slight colicky pain before the bowels are open, but often this is absent. Nutrition gradually suffers, but not to a very marked degree. There can be little doubt that the condition is really a functional neurosis; the intestine shows undue reflex excitability, so that the taking of food into the stomach immediately starts peristalsis of the bowel. Often when the child is brought for medical advice the condition has already lasted several weeks or months, but improvement is generally rapid under suitable treatment.

Treatment.—A mixture of bromides with belladonna given an hour before each meal is very effectual. Small doses of Dover's powder may be used similarly. When the frequency of the evacuations has diminished, liquor arsenicalis (♏ j.-iij.) should be substituted, and may usefully be given with nux vomica.

CONSTIPATION

Both in breast-fed and hand-fed infants constipation is a very common trouble, and often extremely difficult to remedy. In breast-fed infants the cause is usually some defect in the mother's milk, particularly deficiency of fat. Sometimes no fault whatever can be found in the milk, and it seems probable that the trouble is due simply to feebleness of peristalsis, either as part of the general weakness of infancy, or as a congenital condition perhaps the result of defective innervation. It is probable also that the sharp kinks which are so often present with the looping of the long sigmoid flexure in infancy (*vide* p. 88) may hinder the progress of the fæces, and the accumulation of fæces in turn aggravates the kink, so that a vicious circle is established which may account for the obstinate constipation which is not infrequent in infants.

In hand-fed infants, apart from congenital causes, the diet is usually at fault. The mixtures of milk and water which mothers usually give their babies are very deficient in fat, the patent foods are even more so. Sometimes excess of casein seems to cause constipation, but in such cases there is often some catarrh of the bowel, and attacks of diarrhœa may alternate with constipation. A chronic catarrhal condition, such as is common in rickets, is not infrequently a cause of constipation in hand-fed infants, but the rickety constipation is probably due also in part to muscular weakness. Where the stools are pale and whitish the constipation seems to be due to deficiency of bile. Fissure at the anus is said to cause fear of defecation, and so constipation in some cases. Narrowing at the anus or rectum, as a congenital condition, may cause partial obstruction; and more acute obstruction from hernia or from intussusception may occur at any age. As a reflex condition constipation is sometimes a prominent and early symptom of cerebral disease, particularly of meningitis.

Symptoms.—Infants who are costive are often miserable and fretful, they sleep badly, and occasionally strain and grunt; flatulence and colic also are commonly associated with constipation; the temperature may be slightly raised; and where constipation is habitual, nutrition suffers and growth may be delayed. Infants will sometimes strain violently, and quite exhaust themselves in their efforts to defecate, and when the firm fæces are passed they are found to be streaked with blood. As direct results of the straining prolapsus ani and hernia may occur. In two cases under the writer's care piles were present at one month and at eighteen months of age respectively.

Treatment.—This must depend on the cause. If the mother's milk be suspected of poorness she should be encouraged to drink milk freely, and to take a liberal diet with plenty of milk food; it is well if she can take also malt or cod-liver oil. The treatment of the child by drugs given to the mother is usually unsatisfactory, but any of the saline aperients can be used in this way. In hand-fed children the feeding generally requires modification in the direction of additional cream or of diminution—rarely increase—of casein. The addition of a teaspoonful of Mellin's food to one or two of the meals, or the use of malt extract instead of sugar for sweetening, or a little manna (gr. x.-xxx.) given in the milk two or three times a day, may be successful. Sometimes fine oatmeal made into a cream and added to the milk for breakfast, or in infants of ten months or more a drink of beef tea, may be sufficiently laxative. To infants of eighteen months or more a little of the juice of stewed prunes may be given, or well-baked apples; sometimes a little treacle given with oatmeal, or a little honey, will keep the bowels regular. Gentle massage along the course of the colon should be done every morning, and it is most important that the infant should be held over a chamber at a fixed time once or twice a day, even if the bowels fail to act, for in infants as in adults "habit is second nature."

Drugs may be necessary, but the less they are used the better. Saline aperients, sodium phosphate (gr. x.-xv.), magnesium sulphate (gr. v.-x.), or sodium sulphate (gr. ij.-iv.) may be used alone or in combination; sometimes a single dose in the morning is sufficient, sometimes they must be given three times a day. Syrupus cascaræ aromaticus (♏ v.-x.) with sodium sulphate may be tried, but cascara sometimes gripes, and is very unreliable in its action. Where the constipation seems to be due to weakness, minute doses of nux vomica with compound decoction of aloes may be tried. Sometimes a small dose of gray powder given once or twice a day is effectual. Where the stools are pale or putty-coloured, tincture of podophyllum (♏ j.-ij. three times a day) is sometimes serviceable; it may be given in mixture or on powdered sugar. As an occasional aperient, syrup of senna (♏ x.-xv.) or calomel (gr. j.-ij.) is suitable for infants; castor oil should not be used where there is any tendency to habitual constipation, as it tends to make this worse afterwards.

Enemata are often necessary, and for occasional use there is no objection to soap and water or glycerin, but the smallest that will work is the best, for it is possible that large injections may produce some atony of the gut, and so aggravate the constipation. On this account olive oil (½-1 ounce), followed by one or two ounces of soap and water, is better than a large soap and water enema. Where frequent injections are necessary, glycerin is apt to be too irritating to the rectum. The simplest and safest remedy for frequent use is a piece of soap used as a suppository, and for a time this is usually efficient, but if used very often it is apt to lose its effect.

Congenital Dilatation of the Colon

This name has been applied to a rare condition in which obstinate constipation is associated with great dilatation and hypertrophy of the colon, apparently without organic obstruction. The name is perhaps not strictly accurate, for although it seems probable that some congenital abnormality of function or structure underlies this condition, and the chief symptom, namely, constipation, has in most cases been present from birth, the dilatation of the colon cannot generally be detected clinically until some weeks or even months later.

Almost all the cases of "idiopathic" dilatation of the colon which occur in childhood appear to be of this variety, but a very similar condition occasionally begins in adult life, although in some of these cases also there is a history of much constipation from birth.

It seems possible that there may be some family predisposition to this condition, for in one case that came under the writer's observation a brother had died of it; and in another a second child had died apparently with the same condition.

It is a curious fact that almost all the recorded cases have been in boys.

Symptoms.—The child is apparently healthy at birth, but there is great difficulty in getting the bowels open. For a week or more they may not act, and in one case nineteen days elapsed after birth before they were opened. Very rarely constipation does not begin until some weeks or even for two or three months after birth. When the child is a few months old, sometimes only when it is a few days old, the abdomen is noticed to be large, and with increasing constipation the abdomen may become enormously distended. In one case the distension was so great that the heart was pushed up so that the upper limit of cardiac dulness was at the first rib. Œdema of the lower limbs may result from pressure of the distended bowel on the veins.

The most characteristic feature, however, is the visible peristalsis of the colon, which has sometimes attracted even the mother's notice. The surface of the abdomen is seen to rise and fall in irregular eminences and depressions from time to time, corresponding with the slow vermicular movements of the enormous coils of hypertrophied colon which seem to fill the whole abdomen.

Usually in spite of all treatment the constipation persists; the bowels are open only at long intervals, and by artificial means; in two cases under the writer's observation the intervals were sometimes as long as five or six weeks. During these periods there is often headache and vomiting, and the breath is sometimes very offensive. Abdominal pain is usually absent throughout, except sometimes towards the end of the illness.

The appetite in some of these cases is very large, but the child does not thrive; it is poorly nourished, and in the later stages may be considerably wasted.

Sooner or later, sometimes within the first year, sometimes not till the child is ten or twelve years old, the distension of the abdomen becomes extreme, there is some tenderness on palpation, and the constipation is replaced by diarrhœa, the stools being liquid, drab-coloured, or pale yellow, and perhaps frothy. The temperature, which hitherto has been normal, may now rise, and in a few days, partly from respiratory difficulty, owing to the abdominal distension, partly perhaps poisoned by absorption from the dilated intestine, the child dies.

Morbid Anatomy and Pathology.—The large intestine is found to be enormously dilated, and its muscular wall hypertrophied. The dilatation is not always uniform throughout, and it probably varies somewhat during life with the changing relations of the bowel in peristalsis; the hypertrophy, however, seems to affect the whole large intestine down to the anus. Often some recent ulceration of the mucous membrane is present, generally transverse and linear in appearance, the result, no doubt, of the acute and extreme distension which precedes death; the ulceration is seldom extensive, and may be limited to the part where the distension is most marked. The distension of the bowel is found to be due partly to gas, partly to accumulation of fæces which are often quite liquid; but the condition found post-mortem is probably different from that which exists during the greater part of life, for during the last few days or weeks of life, as already mentioned, acute symptoms supervene, and there can be little doubt that these coincide with the occurrence of a superadded acute atonic distension of the bowel, and inflammation and ulceration of the mucous membrane; the pale liquid fæces also are a symptom of this final exacerbation. The small intestine is normal. The most remarkable characteristic in the morbid anatomy of this condition is the apparent absence of any mechanical obstruction. Its pathology is uncertain. It is noteworthy that there is no evidence at present that there is any hypertrophy or dilatation of the colon at birth, the only part of the condition which is actually congenital is the constipation. Any theory which is to explain all the cases must account for the fact that in some cases, at least, the whole large intestine, down to the anus, is hypertrophied. Spasm of the rectum or of the sphincter ani might explain this, but in most cases no such spasm has been found. Kinking at the sigmoid loop, or some abnormal arrangement of the mesentery, fails to explain the affection of the part below the sigmoid flexure. Possibly in some cases there is some congenital defect of innervation of the intestine so that its contractions are ineffectual and constipation results, with secondary dilatation and hypertrophy.

Diagnosis.—The salient points in the diagnosis are the history of constipation from or soon after birth, the presence of visible peristalsis of greatly enlarged coils of intestine, and the absence of any obvious cause for obstruction. This last point is important, for a congenital stenosis of the rectum, or a narrowing of the anus, either congenitally or from an imperfect result after operation for imperforate anus, may produce an almost exactly similar result. Such malformations are readily excluded by examination.

This condition has been mistaken for tuberculous peritonitis; and in the later stage the thin child with its distended abdomen suggests such a diagnosis at first sight; but apart from the history and the dilated coils of intestine, the congenital condition is distinguished by the complete absence of that doughy resistance and the band-like masses which are so characteristic on palpation of the abdomen in tuberculous peritonitis.

The absence of fluid thrill and of the characteristic shifting of dulness with position, taken together with the history, should serve to exclude ascites.

Very rarely in childhood adhesions from past peritonitis cause obstruction of the bowels with distension of the abdomen and visible peristalsis of dilated coils of intestine, but such adhesions are likely to affect the small intestine, and careful inspection will generally detect this difference partly from the size and shape and partly from the position of the coils. Too much importance, however, must not be attached to position, for in congenital dilatation of the colon the huge coils occupy most unusual positions, the sigmoid loop in particular frequently extends across into the right iliac fossa, and may rise above the umbilicus.

Prognosis.—In five cases under the writer's observation the ages at death were respectively 4½ months, 4¾ months, 5 months, 3 years, and 3¾ years, and two others were still living at the ages of 7 years and 10 years. Two cases have been recorded in which the patients survived to adult life, but this is quite exceptional. In almost all the recorded cases death has occurred under the age of 12 years. Whether the condition is ever recoverable may be doubted; certainly under treatment there may be considerable improvement.

Treatment.—In the early stage much can be done to prevent the condition becoming worse. It must be impressed on the parents how important it is that they should never relax their efforts to keep the child's bowels regular. The bowels must be opened at least every two days.

As to the means to be employed to overcome the constipation, careful selection is necessary. Drugs given by mouth too often prove useless, but are worthy of trial, especially in the early stage, before the dilatation is very great. A mixture of liquor strychninæ with sodium and magnesium sulphate may be given three times a day, or belladonna with aloes and nux vomica may be tried. A morning dose of apenta water has acted well. In some cases calomel has seemed to be most useful, and probably even in the later stages has a decided value, not only as an aperient, but also as an antiseptic, where there is a tendency to decomposition and fermentation in the dilated bowel.

Gentle massage of the colon along its course is quite safe, and sometimes decidedly beneficial, in the early stage, but when distension is extreme, especially when the stools are liquid and there is tenderness of the abdomen, the likelihood of ulceration must be borne in mind, for there may be, as Dr. Rolleston and Mr. Haward have suggested, a risk of rupturing the intestine by massage.

In almost all cases drugs given by mouth prove ineffectual sooner or later, and enemata become necessary. For the reasons stated above (*vide* p. 113), olive oil, either alone or mixed with castor oil, followed at once by a small soap enema, is perhaps the least open to objection. In some cases it becomes necessary to clear out the accumulated fæces from the rectum with the finger or with a scoop.

The diet in the early stage may be modified, as in cases of simple constipation; in the later stage it is well to let the diet be as simple as possible, and those articles are to be preferred which leave but little fæcal residue, such as milk, eggs, soup, etc.

At any time an acute exacerbation of distension is liable to occur, especially when the bowels have been allowed to remain constipated for a long period. The likelihood of ulceration at such times must be remembered, and immediate steps must be taken to reduce the distension. Sometimes a turpentine enemata (Ol. terebinth ♏ x., with soap and water ℥vj., for a child of six months) will give temporary relief. Sometimes mere digital examination of the rectum will allow gas and fluid fæces to escape. The passage of a long soft catheter up the rectum may be tried, and, combined with very gentle pressure on the colon, this may evacuate some of the gas. If gas or liquid fæces continue to come away through the tube in occasional gushes it is sometimes well to leave the tube in the rectum for an hour or more to prevent reaccumulation of flatus. Creasote (♏ ¼-j. every three hours) should be given by mouth, as it certainly gives some relief to acute distension.

The question of surgical interference is one of some difficulty. In the early stage the general health is often quite good, and one has to balance the possibility of the child living for several years, perhaps even to adult life, with the use of simple medicinal remedies, against a possible cure by an operation which experience shows to be one of great risk. On the other hand, if surgical interference be deferred until the distension is extreme, the child is in far worse condition to stand the operation, and the difficulties of operation are considerably increased. Some of the cases in which surgical treatment has been tried were in the extreme stage, and the fatal results are perhaps to be attributed in part to this fact. It must be mentioned, however, that in two cases operation appears to have been successful, one in which colotomy was performed (Osler), the other in which the descending colon and rectum were resected (Treves).

Prolapsus Recti

This is a frequent result of straining from any cause. It is more common in children of two to six years than in actual infancy. In infants it is often associated with a marasmic condition, the tissues of the ischio-rectal fossæ are so wasted that they afford insufficient support to the rectum, which therefore tends to prolapse. Constipation, diarrhœa, the irritation of thread-worms, a polypus in the rectum, a tight foreskin, calculus in the bladder—any of these conditions may be the exciting cause of prolapse of the rectum.

Symptoms.—In the mildest cases only a narrow rim of mucous membrane projects through the anus, and the projection may be so slight that at the end of defecation it is pulled in by muscular contraction without artificial aid. Such a degree is of importance only as the precursor of the more troublesome condition, where the prolapsed mucous membrane remains down until returned artificially. In more severe cases two or three inches of extroverted rectum may project from the anus, and in this condition the whole wall of the bowel descends, and even the peritoneal pouch may be dragged down. The prolapsed portion is seen as a cylindrical tumour projecting from the anus, and showing on its outside the bright red, often congested, mucous membrane, and at its apex the lumen of the bowel. Where the prolapse has been allowed to remain down for several hours or days the exposed mucous membrane may become dry and cracked, bleeding on the least manipulation, and sometimes deeply ulcerated.

Prognosis.—As a rule, treatment of the cause produces rapid improvement, the prolapse becomes less frequent, and then ceases altogether. So long as it can be reduced easily, and is never allowed to remain down more than a few minutes, the prolapse is not dangerous; but where it has been allowed to remain unreduced for several days, and reduction has been accomplished with great difficulty, the writer has twice known the condition to prove fatal.

Treatment.—The reduction of the prolapse can usually be accomplished by gentle pressure with the fingers covered by a soft oiled rag, the child meanwhile lying on its side. Application of cold, in the form of iced water, to the prolapsed mucous membrane may assist reduction in difficult cases. Where there is great difficulty, owing to continual straining, or to swelling or inflammation of a neglected prolapse, it may be necessary to administer chloroform. After the prolapse has been returned a starch and opium enema should be given, and for some days astringent injections, or injections of cold water, may be used once or twice a day. The application of a broad strap of plaster across the buttocks, so as to pull the buttocks together and support the rectum, is often effectual, and with this a pad over the anus may be combined. Merely lying on the back seems to do good in some cases. A very efficient method for bad cases is to suspend the legs by stirrups, as is done sometimes for fractured thigh, the child lying on its back with the hips supported on a pillow. In a troublesome case the child should defecate, lying either on its back or on its side, and the buttocks may be supported during defecation by the pressure of a hand placed on either side of the anus. It has been recommended that a chamber-pot with a wooden lid, in which there is the smallest practicable opening, should be used, the lid pressing on the buttocks and so supporting the rectum.

Operative measures are hardly ever necessary for prolapse in childhood. In some cases, however, where it has persistently recurred, the laxity of mucous membrane which predisposes to recurrence has been treated by the actual cautery; this is drawn in three or four vertical lines down the whole length of the mucous membrane on the exposed surface of the prolapsed portion. The application of acid nitrate of mercury has also been recommended (Jacobson). Quite as important as the local treatment is the treatment of the constipation or diarrhœa or other cause to which the prolapse is due; in many cases, indeed, this is all that is required.

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General Paralysis.

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See also PARALYSIS.

SYNONYMS.—Progressive general paralysis; Dementia paralytica; Paretic dementia; General paresis; General paralysis of the insane. *Paralysie générale des aliénés, Folie paralytique* (Fr.); *Paralyse des Irren* (Ger.).

Definition.—General paralysis of the insane is a progressive and incurable disease of the nervous system, characterised by bodily symptoms which end in paralysis, and mental symptoms which end in dementia, the disease running a fairly definite course of development, and terminating fatally in from two to three years.

HISTORY.—Willis is accredited with having first drawn attention to this disease as early as 1672. Haslam and Perfect also seem to have recognised it at the close of the last century. Esquirol noted the extreme gravity of those cases in which dementia was complicated with paralysis, but it was reserved for his pupils, Georget, to describe the condition under the name of "chronic muscular paralysis," Delaye, under that of "incomplete general paralysis," and Calmeil, under that of "paralysis observed in the insane." Up to this time the affection was regarded as a special form of paralysis complicating an already existing mental disease. Bayle (1822) formulated a theory that the condition was not a mere complication of insanity, but a true morbid entity. He insisted on the existence of arachnitis or chronic meningitis as its predominant lesion, ambitious delusions as its necessary symptoms, and completed the picture by a description of its various stages of development.

Parchappe (1838) followed the lead and strove to prove that the affection was a special form of insanity. In 1846, however, there was a slight reaction, and Requin, MM. Sandras, Lunier, and Baillarger endeavoured to restrict this view. They recognised two forms, both progressive—the one with mental perversions, the other without. In 1858 a long discussion ensued between the partisans and opponents of Bayle's views, and the notion of the disease being a morbid entity was accepted. Since then, however, its anatomico-pathological features have been questioned, and an enormous mass of etiological and clinical data have gradually accumulated, with the result that the unitary theory is no longer able to hold its ground.

Ball described it "as a genus comprising many species," and at the present time many recognise that it is not a single malady, but a group of more or less distinct diseases, varying according to their causes or their lesions.

Even the most ardent of the advocates of the entity theory have in their descriptions of the disease included so many factors of causation, so varied symptoms, and so many different types of disease, that the olla podrida of general paralysis is in excess of that of any other disease.

The writer has pointed out (*Journ. of Mental Science*, p. 305, 1896) that before we can decide the question of the existence of true general paralysis, we have to differentiate between its group of symptoms and those of progressive partial paralysis. "From a pathological standpoint general paralysis, as we at present know it, seems to include every known cerebral degeneration as a possible adjunct of its pathology, *i.e.* it includes many affections which often, occurring independently, are in reality special paralyses. From the physical side we have to consider two broad types of disorder, viz. paralyses which are progressive and widespread, and paralyses which tend to progress, but which nevertheless remain, broadly speaking, only partial." From the mental side we have similar difficulties, inasmuch as the abnormal mental manifestations range through every grade of every faculty, thereby rendering a concise, comprehensive, or complete description impossible. The various new clinical facts, such as the remissions and the recognition of latent paralyses, syphilitic, saturnine, alcoholic, and paralytic insanities, have gradually overthrown the entity theory, which includes but fails to explain them. Were we to exclude all those types which simulate progressive general paralysis (pseudo-forms), but which do not fulfil the rôle laid down in most text-books, general paralysis itself—as a distinct disease—would fade into insignificance, or even cease to exist. Until, however, we have carefully differentiated the innumerable factors included in what is at present known as "general paralysis," it is convenient to retain the term, and to use it in describing a paralytic dementia consisting essentially of dementia combined with progressive paralysis.

Causation.—General paralysis of the insane affects most commonly men of middle age whose nervous systems have been unduly exercised in the struggles and competitions of life, and in most cases the disease is further determined by vicious modes of life, errors of diet, injudicious use of stimulants, undue exercise of sexual functions, and neglect of the restorative power of sleep. It is comparatively rare in a purely agricultural population, but among those who dwell in cities and are subject to stress and strain it is very common. It is in the great centres of population, where the people are too thick on the ground, that the constant struggle for survival reacts most upon the nervous systems of a nervous age. It is among the full-bodied, vigorous, and active that the disease finds its abode, and it is the inextinguishable thirst for warm, vital human life, the preference for lit streets and the restless phantasmagoria of the city, that attracts men burning with the fires of a full life. The craving for sensations and satisfactions of many-sided life has its tragic side, and in endeavouring to devour the world men but consume themselves in the process. In brief, general paralysis is the outcome of stress and strain, alcohol and syphilis, all of which are met in the desire for keen, quick, ample life. In every sense it is the mind which is the standard of the man, and nothing succeeds like alcohol and syphilis in effacing the rational and moral faculties of even the noblest and richest.

It is comparatively seldom that alcohol alone is attributed as the cause of general paralysis, but intemperance does not necessarily mean only obvious and palpable drunkenness. From the very moment in which alcohol has disturbed the healthy exercise of the mental faculties, or has impaired the moral sense by unduly exciting the animal passions, or has in any way unfitted a person from discharging his duties in the proper struggle for survival, from that moment has there been guilt of intemperance. A nation must be depraved and wretched in proportion as it abounds in drunkenness, and the ever-increasing weakness and brain decay as manifested in general paralysis is but one indication of the disastrous results of alcohol.

Of late years the relationship of syphilis to general paralysis has been much discussed. The writer's experience has led him to believe that syphilis is one of the most important of the factors of causation of a degenerative neurosis which is included in and described as general paralysis. At least 50 per cent of the cases of general paralysis under his care have had definite histories of syphilis, and probably, if all the facts were known, the percentage would be greater. The following types of insanity associated with syphilis occur:—

1. Types not entirely conforming to the classical descriptions of general paralysis.—The symptoms are: muscular tremors in tongue and hands, alterations in speech, inequalities of pupils, illusions or hallucinations

of the special senses, depression or exaltation, loss of memory, mental weakness, temporary loss of consciousness, and not infrequently partial paralysis or hemiplegia. The diagnosis is difficult, and sometimes an autopsy alone is able to determine the existence of either simple cerebral syphilis or the diffuse periencephalitis of general paralysis. To this type Fournier has applied the name of *general syphilitic pseudo-paralysis*, or general pseudo-paralysis, implying in no respect any morbid entity whatever, still less a "modified general paralysis," and still less "general paralysis" peculiar to syphilis, but merely implying a particular modification of cerebral syphilis recalling more or less the pathological physiognomy of general paralysis.

2. Types which have a history of syphilis, but present no feature pertaining to syphilis, or are exclusively syphilitic, are not influenced by anti-syphilitic treatment, and have a pathology of the nature of a general encephalepathy rather than of cerebral syphilis. The symptoms are mainly those of so-called true general paralysis, and the condition may be termed *parasyphilitic general paralysis.*

3. Types seen in early life and due to inherited syphilis.—Not only may there be arrest or defect of mental or physical development, but there may be epilepsy. In some cases local lesions about the cranium, membranes, or the brain itself may account for the paralysis and the mental defect. There are, however, other cases in which the evidence of syphilis is quite clearly determined. These cases present a remarkable resemblance to general paralysis. The salient features of some of them are slow but progressive dementia, with steady development of generalised paralysis and great emaciation. Whether they are true cases of early general paralysis is still doubtful; they might be termed cases of *congenital parasyphilitic progressive paralysis.*

4. Numerous other types of cases with mental or physical symptoms occur, without, however, bearing any resemblance to progressive paralysis; they need not, therefore, be considered here.

The close relationship of tabes to general paralysis has been urged quite recently by Mott. The relative frequency of the occurrence of syphilis as a causal factor in both diseases is noticeable, but this alone hardly seems to justify the assumption that the two states are identical. Systemic spinal lesions of sensory or motor tracts might result from the same cause, and the proof of syphilitic origin of tabes, primary cerebral spastic paraplegia, or of general paralysis, does not warrant the assumption that they are one and the same disease.

PATHOLOGY.—None of the morbid brain changes found can be said to be distinctive or exclusive to general paralysis. The skull-cap is usually heavier than normal and may exhibit exostoses. The dura mater not uncommonly has rusty discoloration of its inner surface, or arachnoid cysts may be present. The subdural or subarachnoidal fluid is turbid and increased in amount. The arachnoid itself may be thickened, opaque, watery, or gelatinous. The pia, or stripping, adheres to the cortex cerebri along the summits of the gyri, especially of the frontal and parietal lobes. The gyri are atrophied and the lateral ventricles widened. The atrophy markedly affects the central and precentral gyri, basal ganglia, pons, and medulla. Small foci of softening are common in the gray and white matter of the parietal, central, and temporal gyri. Degeneration of the arterioles in conjunction with increase of arterial blood pressure may cause hæmorrhagic foci, especially about the basal ganglia, pons, and medulla, gray degeneration and atrophy of the cranial nerve roots are frequent. The dura mater of the spinal cord may be adherent to the vertebræ, the cord-tunics being thickened chiefly posteriorly. Hæmorrhagic or calcareous deposits on the inner surface are not so frequent as in the brain. Various systemic lesions of the cord itself occur, the blood-vessels are frequently congested, and adhesions may be formed between the pia and cortex.

The histological changes in the brain substances are—increase of blood-vessels, which are distended and tortuous; proliferation of nuclei of the vessel wall, and extravasation of leucocytes; blocking of the subadventitial and perivascular lymph spaces by lymph-corpuscles and waste products; increase of glia-cells, especially in the outermost layer of the cortex; degeneration of the nerve cells and their processes, beginning usually in the apical processes, the cell-body undergoing granular, fuscous, sclerotic changes, or vacuolation; wasting and degeneration of the cortical association nerve-fibres. In brief, the connective tissue becomes hypertrophied and the nerve tissues atrophied. The peripheral nerves, the ganglia of the sympathetic, the muscles, and various organs of the body also exhibit evidences of degeneration. The real nature of the pathological process is as yet undetermined, and we do not know how far it may be attributed to the influence of a toxin derived from without or within.

SYMPTOMS.—1. *Stadium Prodromale.*—Sometimes the disease comes on suddenly without any warning, but usually there are sensory, motor, or mental symptoms. The sensory warnings are, sudden losses or defects of the special senses, illusions, or hallucinations. The motor are, slight or temporary aphasia, hesitation in speech, loss of power of expression, blurring of articulation, tremor of tongue, ataxic or spastic alteration in gait, partial paralysis, convulsive seizures, inequality of pupils, reflex iridoplegia, changes in muscular electric reactions, alterations in handwriting, impairment of highest physical technique. The mental warnings are, loss of highest powers of adjustment of the mind to the requirements of the individual's profession or trade, loss of power of attention, restlessness, changes in temper, moral perversions, and loss of control. General instability, fitfulness, undue emotionalism, boastfulness, expansive benevolence, extravagance, etc. Vaso-motor symptoms, such as congestive attacks, cephalalgia, vertigo, hyperidrosis, local cutaneous hyperæmias, cyanosis, gastro-intestinal disorders may occur, or the trophic functions may be deranged, as shown by changes in the skin, hair, nails, muscles, and bones. The secretions may also be altered in their composition. This stadium has an average duration of one year.

2. *Stadium Acutum.*—Usually ushered in by melancholia, mania, dementia, or by convulsive seizures followed by confusion or stupor. The physical and mental symptoms become more fully developed. This stage usually extends over a period of about a year. The sensory symptoms become more marked in the form of definite illusions or hallucinations; the tinnitus aurium, deafness, diplopia, amblyopia, colour blindness, anosmia, ageusia, parageusia, and the disturbances of general sensation of the stadium prodromale, now become misinterpreted, and form a partial basis for the development of delusions. Melancholia with hypochondriasis may form the chief mental symptoms, or there may be a maniacal outburst with great exaltation, violence and delirium, with rapid advance towards terminal dementia and death. In milder types there may be merely weak-mindedness, with or without exaggerated ideas, or megalo-maniacal delusions. Impulsive acts and emotional outbursts are common. Alteration in character is very marked, the docile become irritable, and the hard facile. Seldom do they recognise the disease from which they suffer, even though they themselves may be medical men and able to diagnose it in others. The exaltation, or *megalomania*, though common in general paralysis, is not invariable nor is it confined to it. The general feeling of benevolence in general paralytics, however, is fairly constant, whereas it is not common in alcoholic mania, delusional insanity, or in monomanias of grandeur. Micromania, or the sense of "belittlement," also occurs, and is usually associated with some perversion of the cutaneous sense.

The *memory* may be altered in various ways. Sometimes in the earlier stages there is hypermnesia or exaltation of memory; in other cases there is a progressive amnesia from the first. The "law of regression" is here exemplified, in that the loss advances progressively from the unstable and most recent to the stable or more organised acquirements. In these cases of general dissolution of the memory an invariable path is followed, viz. memory of recent events goes first, then that of ideas in general, next feelings, and lastly acts. In instances of partial dissolution the loss also follows an invariable path, viz. proper names, common nouns, adjectives and verbs, interjections, gestures. Clinically, it is important to note that progressive loss of memory is usually pathognomonic of cerebral degeneration. In the early stages of general paralysis the impairment of memory is sometimes the most marked symptom. Sometimes the law of regression undergoes a remission, or the progression may be arrested for a time. Progressive loss of memory, however, is not confined to general paralysis, but may exist in syphilitic disease of the brain or meninges; various organic lesions of the brain, meninges, or vessels; in association with idiopathic morbid processes, traumatisms, toxic agents, etc. Sometimes progressive loss follows upon spinal affections, such as locomotor ataxy or multiple sclerosis; or it may result from epilepsy, hysteria, somnambulism, chorea, paralysis agitans, asthma, exophthalmic goitre, or myxœdema. Various forms of paramnesia are also common in general paralysis.

The special senses may in the earlier stages be exalted and hyperacute, but as the disease progresses they gradually become blunted and defective. Hallucinations of sight and hearing are present at some period in quite 50 per cent of the cases. Anæsthesia of the cutaneous nerves is almost invariable in the later stages. Analgesia is also a noticeable symptom. Ulnar analgesia (tested by pressure on the trunk of the ulnar nerve in the ulnar groove) is more frequent in general paralysis than in other forms of disease, even than in those which have other severe disturbances of sensibility. This sign (*ulnar sign*) is most common in tabetic forms of general paralysis. The absence of reflex action, when the finger is thrust between the fauces (*pharyngeal sign*), is also very commonly met with.

The eye symptoms are numerous and of great significance. Transitory defect of vision may form an early symptom, later there may be optic neuritis, atrophy, and blindness. The *pupils* are generally unequal, often irregular in outline and defective in their reaction. Inequality, though common, is not pathognomonic of general paralysis. The reaction of the retino-iridal apparatus to stimuli is, however, of great importance. The rhythmic oscillations of the iris corresponding to the respiratory rhythm may be abolished; the normal dilatation of the pupil associated with bodily muscular movements may also be absent. Of more importance, however, is the failure to produce dilatation by acoustic, tactile, painful, and electrical stimuli. This "reflex dilatation" (Erb) may exist in spite of anæsthesia of the surface stimulated, and the failure of this reaction in cases of general paralysis is unaccounted for. It is difficult to obtain this reflex when the pupils are contracted. Sometimes, however, though no response can be obtained to stimulation of the skin, dilatation may result from emotional disturbance through the agency of the sympathetic. The so-called "*hippus*," or wide rhythmic oscillations occurring independently of respiration, is a symptom also seen in general paralysis. Consensual dilatation or contraction to light stimuli is sometimes impaired. Failure of one eye to react both to indirect and direct light stimuli indicates that eye to be the one affected, and the lesion to be a paralysing lesion affecting the sympathetic fibres of the defective side. When there is no marked contrast on shading both eyes, failure on one side to contract or dilate on direct light stimulation, failure on the same side to excite its fellow consensually, and its own consensual reaction, following excitation of the sound eye, then the lesion is probably in front of the chiasma, either retinal or affecting one optic nerve.

Bevan Lewis has indicated that in certain cases of general paralysis the initial contraction to concentrated light may be succeeded by a secondary dilatation—the pupil expanding widely in spite of the concentrated illumination, and he regards this as one of the earliest signs of a failing motor oculi nucleus—the centric expansions of the third nerve being readily exhausted.

There seems to be no definite relationship between pupillary inequality and the efficiency of the iridal reactions. Paralytic mydriasis is usually associated with exaggerated knee-jerks, and sometimes ankle clonus. Tongue tremors, marked affections of articulation, tremors of the lips and muscles of the lower facial group and of the hands. Tremors and twitching movements of the muscles of the brow are said to indicate alcohol as a probable factor of causation. Tabetic cases with iridoplegia have also altered reflexes. Romberg's sign may be present. Sometimes this sign cannot be obtained upon closing the eyes and approximating the feet, but if the body is bent forward swaying movements occur. Where the pupils are small or myotic the symptoms are usually tabetic. Accommodative reaction may be retained, but the Argyll-Robertson symptom is present. Usually the oculo-motor symptoms appear first—the loss of knee-jerk appearing later on.

Bevan Lewis tabulates the following five groups of symptoms as occurring in progressive paralysis of the insane:—

1. Paralytic mydriasis; a partial reflex iridoplegia (light). Increased myotatic irritability. Excessive facial tremor and speech troubles. Great optimism with profound dementia.
2. Mydriasis with associative iridoplegia rapidly passing into the cycloplegic form—an early symptom. Frequent myotatic excess, but no contractures. Late speech trouble. Acute excitement with frequent convulsions. Very rapidly fatal course. (Preponderance of syphilitic history.)
3. Spastic myosis; a complete reflex iridoplegia. Absent or greatly impaired knee-jerk. Failure of equilibration; locomotor ataxy, defective sensibility. Very defective articulation. Much optimism and excitement.
4. Late eye symptoms: paralytic mydriasis, a partial reflex iridoplegia (for light only). Ataxic paraplegia confined to lower extremities. (Arms do not participate.) Great facial ataxy with extreme troubles of speech. Epileptiform seizures ushering in pronounced mental enfeeblement.
5. No oculo-motor symptoms beyond occasional inequality. No contractures, but notable myotatic excess. No disturbance of equilibration, locomotion, or sensation. Speech troubles not pronounced. Epileptiform seizures very rare, but from the first progressive deepening dementia.

The handwriting is almost characteristic. Letters are often shaky, incomplete, detached, or illegible. In the early stages general paralytics also often write voluminously and quickly.

Seizures of various kinds occur either as early symptoms or as marked features of the later stages of the disease. They vary in severity from simple congestive to epileptiform or apoplectiform attacks. Some have merely slight attacks of giddiness, temporary loss of consciousness (*petit mal*), or transient loss of function of one of the special senses. In the stadium acutum congestive seizures are common and may resemble those of epilepsy or apoplexy. These attacks sometimes come on without warning, or they may be heralded by slight rise of temperature, confusion of ideas, or by some gastric disorder. They are irregular in their recurrence. Sometimes the epileptiform fits follow a definite line of development, and they usually leave behind them more physical and mental weakness than do ordinary epileptic fits. Apoplectiform seizures, on the other hand, are followed by less serious physical and mental troubles than in the case of true apoplexy. The apoplectiform seizures may be attended with one-sided convulsions, with rigidity, convulsive movements, turning of the head to one side, and coma lasting for hours. They resemble apoplexies, except in that the condition is temporary, and any paralytic effects usually disappear in the course of a few days. Sometimes, however, mental dulness, drowsiness, or excitement follows the apoplectiform attacks, and may persist for days or weeks. Severe seizures may be followed by gradually increasing coma, palsy, and death. In fatal seizures associated with hemiplegia the brain may be affected by coarse disease or hæmorrhage. Tetaniform and hysteriform attacks also occur occasionally in general paralysis. The temperature is subject to irregular fluctuations quite independent of seizures. In maniacal states the temperature may rise slightly. Sometimes also in hypochondriacal forms there may be an evening rise. If excitement is protracted for several days there may be a rise, followed by a fall with the subsidence of the excitement. In some instances the temperature rises to 102° or 103° without any apparent physical cause. Subnormal temperatures are not common during the stadium acutum, but in the third stage they are relatively frequent. Fluctuations are relatively more frequent in patients with seizures, excitement, or with motor disorders. The circulation and pulse vary considerably, and in some cases indicating a primary cardiac enfeeblement. In maniacal cases with motor excitement the blood-pressure tends to become lowered: in quiet cases, on the other hand, it is raised. In the stadium acutum the pulse rate is relatively more frequent than is the case in the other stages.

The alimentary functions are usually active and well sustained throughout the course of the disease. During the early stages there may have been gastro-intestinal troubles, but in the stadium acutum the appetite usually becomes increased; organic sensations of discomfort seldom occur; sensations, which in the healthy usually indicate the condition of the stomach and intestines, are absent, and the want of knowledge as to whether they have had enough food leads to great increase in bodily weight. Progressive dysphagia is the rule, and is due to defective reflex action, incoördinate action, and later, paresis of the muscles of deglutition. Sometimes swallowing is almost impossible owing to bulbar lesions, or to pharyngeal and laryngeal anæsthesia. The dangers of death from choking are great, and careful attention to proper mincing of food is necessary.

The functions of the bladder and rectum become affected as the disease advances. The urine is passed involuntarily or retained, or there may be retention with incontinence due to an overfull bladder. The habits become foul, and the patient requires much personal attention. The sexual power, which may have been much exalted during earlier stages, now becomes impaired, and later lost.

The disorders of muscular and motor control are numerous. The lines of expression on the face become obliterated, and there is tremor of the muscles when movements are performed. This tremor is most marked about the muscles of the lips and tongue, and may be finely fibrillar or coarsely ataxic. The speech is difficult, hesitating, blurred, with slurring over labials, sibilants, or dentals. Words are jumbled together, or there is elision of syllables, or a species of stammering. These speech disorders may be due to defects of (1) *ideation*—either defective memory, or impaired association of ideas; defects of (2) *articulation*—ataxic or amnesic; or defects of (3) *phonation*—the vocal cords or the physical apparatus serving them being impaired.

Other symptoms are common, such as slavering, smacking the lips, sucking movements, grinding the teeth, or constant swallowing.

The gait may be ataxic, paretic, spastic, or the attempt to move may call forth trembling of the limbs. In some cases coarse trembling of the whole body occurs, when Romberg's test is attempted. Charcot's dictum that immobility is the finest movement of a soldier is here exemplified, and the co-ordination requisite for immobility is defective. Convulsive tremors without pyrexia, athetosis, and choreiform movements are sometimes seen.

The reflexes, superficial and deep, vary considerably. Exaggeration of knee-jerk is often associated with jerky, tremulous hand grasp, twitching of face, lips, tongue, and limbs, rigidity of proximal muscles of trunk and limbs, seizures, and dementia. Absence of knee-jerk is often associated with coarse ataxic gait, gross disturbances of sensation, cutaneous and articular, with consequent loss of so-called muscular sense, and megalomania.

Various vaso-motor symptoms occur, such as capillary congestions over the malar bones, erythematous and sudaminal affections of the skin, bed-sores, *tache cerebrale*, punctiform hæmorrhages under the skin after seizures, purpuroid blotches, bruise-like swellings, independently of any traumatic cause, othæmatomata, bullæ, eschars, unilateral local sweats, etc. Nutritive and trophic defects are evidenced by alterations in the body weight, dull, earthy, greasy, herpetic, ecthymatous, furfuraceous, or pigmented skin. Herpes zoster is common, and pemphigus parasyphiliticus occurs in the later stages of the stadium acutum. Furuncles and carbuncles appear. Bed-sores occur from pressure and excremental irritation, and assume a chronic type, or they may be acute and due to cerebral or spinal lesions, and appearing mostly after seizures.

The muscles sometimes undergo progressive atrophy or fatty degeneration, or exhibit hæmatomata or extensive ecchymoses as the result of slight injury, etc. The bones tend to become more brittle and more easily fractured. Special arthropathies also sometimes occur.

Remissions of the symptoms may occur during the earlier stages of the disease. These remissions are most frequent in cases with maniacal excitement, and in which alcohol has been a factor in the causation. The remission may be complete, so that the patient may resume work for a time. Seldom is there more than one complete remission.

The *stadium dementiæ* is the third and final stage, and extends over a period of an average duration of one year. The dementia becomes more marked. There is loss of special and common sensation; objects, persons, and places are no longer recognised; the condition is almost purely negative, and the patient is incapable of self-care; occasionally there may be traces of former hallucinations or delusions, or brief attacks of excitement occur. With the mental hebetude there is neglect of the daily wants of nature; vesical and rectal troubles appear; ataxia gives way to paresis, and the patient becomes helpless and bed-ridden. The circulatory and respiratory functions are affected, and dyspnœa and syncope from heart failure are common. Tremors, spasms, atrophies, contractures, teeth-grinding, decubitus, and dysphagia become more pronounced, and death occurs through intestinal, renal, or vesical inflammations, bed-sores, septic infection, congestive seizures, or by œdema of lungs, pneumonia, embolism, or cerebral effusions. The duration of a typical case is therefore about three years. In galloping cases, however, death may ensue within a few months of the onset. In some tabetic cases the duration is of great length, and may extend over six or seven years. Some of the syphilitic and parasyphilitic cases also run through a protracted course.

The DIAGNOSIS is often extremely difficult in the earlier stages. When we remember that the progressive paralysis may start with almost any form of mental or physical disorder the difficulty can be readily appreciated. Careful attention to any history of syphilis or alcohol in the case, and the presence of any of the physical symptoms already enumerated, will aid in coming to a decision. A differential diagnosis will have to be made from alcoholic disorders, which may simulate general paralysis in almost every particular, but from which the patient may recover; syphilitic cerebral affections, already considered; lead poisoning; convulsions of kidney disease; cerebral tumours; tabes; various systemic spinal lesions, with associated mental symptoms; disseminated sclerosis; paralysis agitans; sunstroke; epilepsy; apoplexy; various pseudo-general paralyses, which form sequelæ to fevers, malaria, traumatisms, etc.

Forms of General Paralysis.—The following clinical types are recognised:—(1) Acute or galloping general paralysis. The disease runs a very rapid course, and may terminate fatally in from two to three months from the onset. When the disease is ushered in by acute delirium there may be rise of temperature; physical and mental dissolution proceeds rapidly, and death ensues from exhaustion. Fits are not common. (2) Ordinary general paralysis, with mania and exaltation of ideas. Usually preceded by a period of depression, or hypochondriasis. Maniacal symptoms, with expansive benevolence and a feeling of well-being, supervene. The later stages show nothing characteristic to this group. (3) Melancholic, hypochondriacal, or stuperose general paralysis. Sometimes difficult to detect, but careful attention to the history of the case, syphilis as a factor of causation, and the presence of physical symptoms indicative of paralysis, render a diagnosis possible. Sometimes the depression passes off and mania supervenes. Remissions are not so common as in the maniacal forms. Fits of various kinds may occur. (4) General paralysis of the double form, or circular general paralysis. The first symptoms may be maniacal, followed by remission; next they may be melancholic and again followed by remission. These attacks may alternate and recur several times before a definite diagnosis can be arrived at; but sooner or later the physical symptoms show themselves and the disease progresses to a fatal end. (5) Progressive dementia with general paralysis. The mental symptoms are those of dementia from the outset. There is progressive enfeeblement of all the mental faculties. Fits are common. From being dull and heavy they become fat and fatuous, and in some cases without any maniacal or melancholic symptoms during the course of the disease. (6) Spinal general paralysis, that is, beginning with symptoms of systemic spinal disease. Tabes dorsalis, spastic paraplegia, or disseminated sclerosis may precede general paralysis by several years. Sometimes also general paralysis may be preceded by peripheral neuritis. (7) Developmental or adolescent general paralysis. Sometimes difficult to distinguish from disseminated sclerosis. Parental syphilis is almost invariable. These have already been referred to as cases of congenital parasyphilitic progressive paralysis. (8) Senile general paralysis. Often difficult to distinguish from senile dementia, with brain decay. The physical symptoms are seldom so characteristic of general paralysis as are the mental symptoms.

The physical signs are those most relied upon. The presence of excitement, exaltation, euphoria, and expansive benevolence, together with the characteristic affections of speech, fibrillary trembling of the muscles of the lips, tongue, face and limbs, spasmodic, convulsive, paralytic seizures, alterations in the habits mental and physical, form a clinical picture sufficient to justify a diagnosis. It is seldom that the disease can be diagnosed from one symptom alone; in fact it is often only when the mental and physical signs form a definite clinical *tout ensemble* that the nature of the disease is evident. Much information can be obtained from the patient's friends as to alterations in his habits or modes of life. Before making a diagnosis the greatest care should be taken to ascertain whether alcohol has been a causal factor.

Prognosis.—The result of the disease is inevitable, but its course is variable. In some rare instances the disease becomes arrested, and the condition may remain stationary for many years. Undoubtedly general paralytics live longer in asylums than in their own homes. The relation of fits to the course of the disease is often interesting. Sometimes the seizures hasten the progress, or, on the other hand, they may cause an arrest or even remission. Some of the most rapid cases have had no seizures at all, and it is also noteworthy that fits of slight severity are sometimes associated with rapid progress of the disease. During the later months much will depend upon the amount of personal attention paid to the organic and nutritive functions.

Treatment.—Some cases of simple progressive dementia may be treated at home under suitable nursing and supervision. When there is excitement, marked depression, or certifiable delusions, however, it is advisable to send the patient to an asylum. Change of air or sea voyages are seldom beneficial. A simple life, with plenty of sleep and rest, moderate exercise, plain food, and attention to the bodily functions, are all that can be advised for many cases. Medicine and surgery can avail but little in staying the progress of the disease. For sleeplessness, paraldehyde in drachm doses proves of most service.

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The *Female Generative Apparatus* consists of the Internal Genitals, including the Uterus, Ovaries, Fallopian Tubes, and Parovarium, which lie within the Pelvis; the external Genitals, which are situated in the inter-femoral space; and the Intermediate Organ, the Vagina, which connects together the Internal and the External Organs. The internal organs mainly rest on the inner aspect of the musculo-membranous diaphragm, which constitutes the pelvic floor; the external organs lie on its outer aspect; while the intermediate organ divides the floor into two segments, an anterior and a posterior. So closely are all these structures associated, that it is found to be convenient to consider the pelvis, osseous, ligamentous, muscular, and membranous, along with the organs of generation in the strict sense of the term.

THE OSSEOUS PELVIS.—(1) *Constituent Parts.*—The osseous pelvis is composed of four bones: a pair, the ossa innominata, and two single bones, the sacrum and coccyx. The innominate bones, with the assistance of the soft structures of the anterior abdominal wall, form the lateral and anterior walls of the pelvis; the sacrum and coccyx make up the posterior wall; in its osseous state the pelvis (or bason) is without a floor; this is supplied by the soft parts of the pelvic floor. Each of these four bones is composite, and was originally developed from several parts, which later on fused into one. Each os innominatum consists of three parts: an upper or iliac, an inferior or ischial, and an anterior or pubic, and these meet at the acetabulum, and become fused about the period of puberty; the sacrum consists of five segments, really five vertebræ, and these fuse a year or two later than the parts of the os innominatum; and the coccyx consists of four or five portions, really aborted vertebræ, which do not entirely fuse till late in reproductive life (thirty-five to forty years). About the same age, or later, the coccyx becomes firmly anchylosed to the sacrum, so that there is then one bone, sacrum-coccyx; up to this time the coccyx, which normally is directed forwards, can be pushed backwards, a movement known as retropulsion. The sacrum is jointed to the two innominate bones at the sacro-iliac joints or sychondroses, while the innominate bones meet by means of their pubic portions at the symphysis pubis. The whole pelvis is articulated to the lumbar part of the spine at the sacro-vertebral joint, where the upper part of the sacrum projects distinctly forwards in the promontory, while it is attached to the lower limbs at the acetabula or cotyloid cavities.

(2) *Osseous Pelvis as a whole.*—The pelvis as a whole has the form of an inverted truncated cone slightly flattened from before backwards; the base looks upwards and forwards, and the apex looks downwards and backwards. In its interior is a curvilinear, irregular canal, the upper part of which constitutes the large or false pelvis, and the lower the small or true pelvis. Its external surface may be generally described as roughened for the attachment of muscles, while its internal surface is smooth. The *false* pelvis may be more accurately defined as the part lying above the plane of the brim, bounded behind by the last two lumbar vertebræ, at the sides by the iliac bones, and anteriorly by the non-osseous abdominal wall. The plane of the brim in its turn may be defined as an imaginary surface, bounded behind by the sacro-vertebral promontory and the sacro-iliac synchrondoses, at the sides by the ilio-pectineal line, and in front by the upper border of the pubic bones and of the symphysis pubis. In the adult the normal non-pregnant genital organs do not lie in the cavity of the false pelvis; but in the fœtus and infant the upper part of the uterus and the annexa lie above the plane of the brim, and in pregnancy, after the fourth month, there is a similar but more marked ascent of the internal genitals. In the lateral walls are the internal iliac fossæ, which have a normal depth which it is difficult to express in figures, but which ought to be impressed upon the memory in order to distinguish it from the deepened state of the fossæ in the malacosteon deformed pelvis, and from the

flattened out condition in the rachitic pelvis. There are two important diameters in the false pelvis: the interspinous, which joins together the two anterior superior iliac spines, and measures 24 cm.; and the intercristal, which passes between the widest apart portions of the iliac crests, and measures 27 cm. It is therefore convenient to remember that normally the intercristal diameter is slightly more than one inch longer than the interspinous. The inter-trochanteric diameter is about 31 cm.

The *true* pelvis contains the generative organs, and requires a more detailed description than the false, for in it the phenomena of reproduction take place. It forms an irregular canal slightly enlarged at the middle, *i.e.* irregularly barrel-shaped; it is curved in the same manner as the anterior aspect of the sacrum, concave anteriorly. Its parts, diameters, inclination, planes, and axes call for description. It has three *parts*—brim, outlet, and cavity. The brim is the upper end of the canal, and is also known as the upper strait or pelvic isthmus, and has of course the boundaries of the plane of the brim (*vide supra*). In form it may be called oval or elliptical, but it is most correctly termed heart-shaped or cordiform, for the projection of the sacral promontory indents the oval posteriorly. The outlet of the true pelvis is also called the lower or perineal strait, and it is bounded behind by the tip of the coccyx, at the sides by the ischial tuberosities, and in front by the lower margin of the pubic bones and of the symphysis pubis. Its circumference is broken postero-laterally by the deep sacro-sciatic notches; these, however, are converted into foramina by the sacro-sciatic ligaments, and then the outlet has an ovate form, the broad end being posterior, and being indented by the projection forward of the tip of the coccyx. Anteriorly the circumference is broken by the pubic arch, which has a height of 5 cm. in the middle line, and a width varying from 3 cm. in the upper part to 9 cm. in the lower. Between the inlet and the outlet of the pelvis lies the cavity. It has a somewhat circular form. Its anterior wall has in the middle line a height of nearly 2 inches (4 to 5 cm.), and consists of the posterior aspect of the symphysis pubis and of the pubic and ischial bones as far back as a line dropped vertically downwards from the ilio-pectineal eminences. In this wall are the two obturator or thyroid foramina. The posterior wall is made up chiefly of the anterior curved surface of the sacrum, and has a height of about 6 inches (15 cm.) if the curve be followed. It is worth noting that on this wall, at the junction of the first and second segments of the sacrum, there is often a slight projection which might possibly be mistaken for the promontory which lies above it. The anterior boundaries of the posterior wall are lines dropped from the sacro-iliac synchondroses towards the insertion of the sacro-sciatic ligaments into the coccyx. Between the anterior and posterior walls lie the lateral walls, which have a height of about 4 inches (9 to 10 cm.). The anterior part of each lateral wall is called the anterior inclined plane; it looks backwards, inwards, and upwards, and corresponds to the internal aspect of the bone bearing the acetabulum, and of the body of the ischium and ischial tuberosity. The posterior part is called the posterior inclined plane; it looks forwards, inwards, and upwards, and corresponds to the internal aspect of the ischial spine, and of the sacro-sciatic ligaments and foramina. These inclined planes must on no account be confounded with the planes of the pelvis to be described below; the former have been supposed to play an important part in the mechanism of labour; the latter are geometrical conceptions useful in describing the mechanism of labour.

The *diameters* of the pelvis have a very considerable obstetric importance, and are described in relation to the brim, outlet, and cavity. The diameters at the brim are specially worthy of note; they are six in number—antero-posterior, transverse, two diagonals, sacro-cotyloidean, and sacro-pectineal. The antero-posterior is also called the sacro-pubic, sacro-suprapubic, or the conjugate (d. conjugata vera); it passes from the middle of the promontory of the sacrum to the middle of the upper border of the symphysis pubis, and it measures from 11 to 11·5 cm. The transverse diameter at the brim passes between the widest apart portions of the ilio-pectineal lines, and measures 13·5 cm. The diagonal or oblique diameters of the brim are two in number, right and left; the former passes from the right sacro-iliac synchondrosis behind to the left pectineal eminence, while the left passes from the left sacro-iliac behind to the right pectineal eminence in front; each measures 12·5 cm., but strictly speaking, and on account of the slight physiological deviation of the pelvis to the left side, the right oblique is slightly longer than the left. The oblique diameters, it will also be noted, connect together four bony landmarks, which have been called the cardinal points of Capuron, and so divide the brim into quadrants. The sacro-cotyloidean diameter is sometimes referred to; it passes from the middle of the sacral promontory to the point on the inside of the pelvis which corresponds to the upper and posterior part of the cotyloid cavity on the outside; there is therefore one on each side, right and left, and it measures 9 cm. The sacro-pectineal or diameter of Burns passes from the middle of the promontory to the crest of the pubes immediately above the obturator foramen; it has a length of 10 cm. It is useful to remember that the shortest antero-posterior diameter of the pelvis is not the conjugata vera, but one drawn from the middle of the promontory to the nearest point on the posterior surface of the symphysis pubis, a point which is usually about 5 mm. below the upper margin; this is called the diameter minima; and its length is from 10·5 to 11 cm. The pelvic diameters at the outlet are four in number: antero-posterior, transverse, and obliques. The antero-posterior passes from the tip of the coccyx to the lower border of the symphysis pubis, and measures with the coccyx pressed backwards, 12 cm.; with it in its usual position, 10 cm. The transverse diameter is the distance between the tubera ischii; it measures 11 cm. The obliques are difficult of exact definition, and they have a varying length in parturition, as the sacro-sciatic ligaments yield a little to the fœtal head passing through the outlet. In the cavity of the pelvis various diameters may be measured, but the following are those of most interest. There is the antero-posterior drawn from the third sacral vertebra to the middle of the posterior surface of the symphysis, and measuring 12·5 cm.; there is the transverse, passing between the areas of bone on the inside of the pelvis corresponding to the acetabula on the outside, measuring 12 cm.; there is another transverse diameter which is sometimes of importance—the bi-ischial, the distance between the ischial spines, measuring 10 cm.; and, finally, there are the obliques of the cavity, right and left, passing from the sciatic foramen on one side to the obturator foramen on the other, and measuring about 13·5 cm. (Fig. 1).

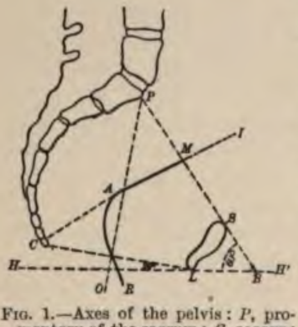

Fig. 1.—Axes of the pelvis: *P*, promontory of the sacrum; *C*, coccyx; *S*, symphysis pubis; *I*, umbilicus; *PS*, plane of the superior strait; *CL*, plane of the inferior strait; *HH'*, line of the horizon; *IMA*, axis of the superior strait; *AR*, axis of inferior strait; *MAR*, curve of Carus; *PBL*, obliquity of superior strait; *CLH*, obliquity of the inferior strait. (After Dorland.)

In addition to the diameters which have been described there are two others called conjugates, which have a very considerable clinical importance, for they can be measured during life. These are the diagonal conjugate or sacro-subpubic diameter, and the external conjugate or diameter of Baudelocque. The former is drawn from the middle of the promontory to the lower border of the symphysis pubis, and measures from 12 to 12·5 cm.; from it the conjugata vera can be deduced by the subtraction of 2 cm. The external conjugate is measured from the spine of the last lumbar vertebra to the upper border of the symphysis pubis, and in order to get the right spinous process in the living subject it is well to draw a line connecting the depressions representing the posterior superior iliac spines and the second spinous process, above this is that of the fifth lumbar vertebra. This diameter is 18 cm. in the osseous pelvis, and 19·5 cm. in the living woman.

By the *inclination* or *obliquity* of the pelvis is meant that it is not in the axis of the body, but placed obliquely to it, as is shown by the fact that in the erect posture the sacral promontory is 9 cm. above the upper border of the symphysis pubis, while the tip of the coccyx is about 2 cm. above the level of the lower margin of the symphysis. The degree of the inclination of the inlet is found by measuring the angle which the antero-posterior diameter of the brim when prolonged makes with the horizon; it is about 60°; in the same way the inclination of the outlet is 10° or 11°, but when the coccyx is pressed backwards the antero-posterior diameter corresponds with the horizon. In the recumbent posture with the sacrum raised the pelvic inclination is 25°; in the same posture with the legs hanging freely over the edge of the table the greatest inclination is obtained (about 10°).

Planes of the pelvis are often referrred to in descriptions of the mechanism of labour. The plane of any part of the pelvis is a surface supposed to touch all points in the circumference of that part; thus the plane of the brim is an imaginary surface touching all points of the circumference of the inlet. It is thus possible to draw a large number of such planes in the pelvis, and the form of each is represented by the relative length of the various diameters thereof. The plane of the inlet has an inclination of 60°, that of the outlet is nearly horizontal. It is noteworthy that the plane with the smallest diameter, that between the ischial spines, is not at the outlet but just above it; it is that between the apex of the sacrum and the lower border of the symphysis; its antero-posterior diameter also does not vary, for the sacrum cannot be pressed backwards as can the coccyx. The *axes* of the pelvis are lines drawn perpendicularly to the centre of the planes, and there are in consequence as many axes as there are planes. The axis of the inlet is directed upwards and forwards, and corresponds with a line passing through the umbilicus and the lower end of the coccyx. The axis of the outlet is directed downwards and slightly backwards. If the axes of the brim, outlet, and cavity of the pelvis be joined together, the result will be a curved line, and this is called the central line, the guiding line, or the line of direction of the pelvis; it indicates the curve which the fœtal head follows in its descent through the pelvic canal in labour (Fig. 1).

Pelvic Differences due to Sex, Age, and Race.—The sexual differences in the osseous pelvis are early impressed upon it, as Fehling pointed out some years ago, and as Arthur Thomson has recently shown. According to the latter (*Journ. of Anat. and Physiol.* xxxiii. 359, 1899) the essential sexual differences are as well defined during fœtal life as they are in the adult.

These are briefly as follow:—In the female the walls are thinner and their bony prominences are less marked; the vertical dimensions are less, the transverse dimensions are greater, and so is the antero-posterior diameter in the cavity; the acetabular cavities are further apart, and the false pelvis is wider, while the true pelvis is more rounded; the pelvic inclination is greater; the promontory is less projecting, and the sacrum is broader and shorter; the obturator foramina are larger, and triangular rather than oval; the pubic arch is broader and more rounded in its upper part, the angle being 90° to 100° instead of 70° or 80°; the depth of the symphysis is less; and, finally, the sacro-coccygeal joint is more movable than in the male. In order to contrast the age differences of the pelvis the fœtal may be compared with the adult female. In the former the promontory is high, the pelvic inclination being 75° to 80°, and the sacrum and coccyx are straight; in the latter the promontory is not so high, it projects forward more, the pelvic inclination is from 55° to 60°, and the sacrum and coccyx are concave; in the fœtus all the diameters are relatively smaller in the true pelvis than in the adult, and the transverse at the brim is only slightly longer than the conjugate instead of being markedly so as in the adult; it is usually stated that the sacrum in the fœtus is relatively narrower than in adult life, but Arthur Thomson (*loc. cit.*) denies this; and, finally, the ischial tuberosities are nearer to each other at the outlet in the fœtal pelvis than are the ischial spines, while in the adult the spines are nearer than the tuberosities. The racial differences of the pelvis have not yet been fully worked out; but it would seem that among the primitive races of America and Australia, and in the case of the Malays, the diameters at the pelvic brim approximate more closely to each other, so that the antero-posterior and the transverse come to have nearly the same measurement. In other words, the inferior races have a pelvis with a more rounded inlet. In addition to the sexual, age, and racial differences which have been described, it must be remembered that pelves of the same age, sex, and race may nevertheless show peculiarities which cannot be called pathological, and must

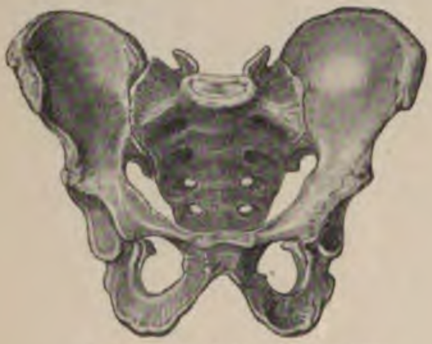

FIG. 2.—Male pelvis seen from the front.

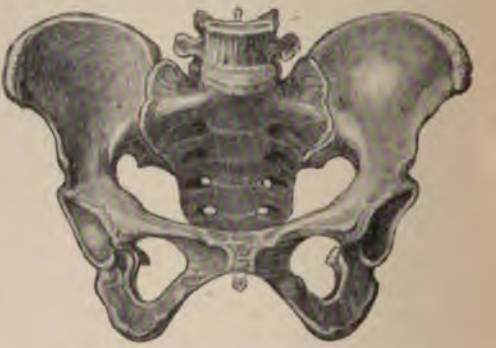

FIG. 3.—Female pelvis seen from the front.

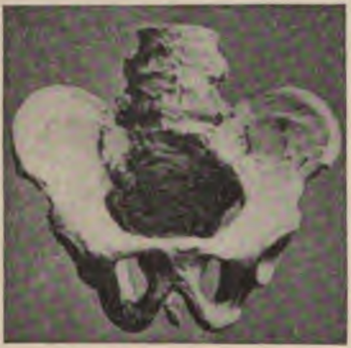

FIG. 4.—Pelvis of seven months' fœtus. (After A. Thomson.)

be regarded as individual; no two pelves probably are exactly alike. Further, it is generally found that the pelvis is not symmetrical, but shows sinistroscoliosis, the symphysis being drawn towards the left (Figs. 2, 3, 4).

THE LIGAMENTOUS PELVIS.—The four bones which make up the osseous pelvis are articulated to each other at the two sacro-iliac synchondroses, at the sacro-coccygeal joint, and at the symphysis pubis. These joints are amphiarthroses; but the symphysis pubis has by some been considered as a mixed joint, arthrodial behind and amphiarthrodial in front, while others have looked upon the sacro-coccygeal as entirely arthrodial. The sacro-iliac articulations, consisting of the auricular surfaces of the sacrum and innominate bones, are held together by four ligaments (anterior, superior, inferior, and posterior); but the great and small sacro-sciatic ligaments which pass from the sacrum and coccyx to the ischial bones also play a part in maintaining the apposition of the articular surfaces. The synovial membrane is small in extent; and the amount of movement possible at the sacro-iliac joints is very limited, but is slightly increased in pregnancy, and is also greater if the symphysis pubis be divided. The symphysis pubis has considerable importance at the present time through the revival of the operation of symphysiotomy. The articular surfaces are oval, and between them is an interosseous fibro-cartilage; they are held together by anterior, posterior, superior, and inferior, or subpubic ligaments. A slight degree of movement is possible at this joint, which is increased if the pubic bones be divided through the obturator foramina; on account of slight swelling and increased moisture of the tissues there is also more marked mobility in pregnancy. An interosseous fibro-cartilage and peripheral ligaments are met with in connection with the sacro-coccygeal joint, which has a considerable range of mobility up to mid-life; but it has to be borne in mind that some movement also occurs between the segments of the coccyx which are united by rudimentary amphiarthroses. Indeed, some writers are of opinion that the movement backwards of the coccyx which occurs in labour really takes place at the first inter-coccygeal joint. The pelvis as a whole is attached to the lumbar part of the spinal column by a median symphysis or amphiarthrosis and by two lateral arthrodia; it is articulated to the lower limbs at the hip-joints; but these joints do not require special description here. The other ligamentous structures of the pelvis are the obturator membranes and Poupart's ligaments: the former close in the obturator foramina and complete the anterior pelvic wall; the latter stretch from the anterior-superior iliac spine to the spine of the pubic bone on each side, and the part of it which is attached to the pubic crest is called the ligament of Gimbernat.

Internal Generative Organs.—The internal female genital organs include the ovaries, the Fallopian tubes, and the uterus; and along with these may be described the parovarium. The organs will first be considered separately, and later their relation to the soft structures filling up the pelvic outlet will be taken up.

OVARIES.—The ovaries are two small bodies of an ovoid form lying one on each side of the uterus and having the function of forming ova. They are the distinctive organs of the female. They are situated at the level of the pelvic brim and are attached to the posterior surface of the broad ligament of the uterus by the anterior border alone, and not, as has sometimes been stated, by the anterior surface. Their exact position and the direction of their long axis vary slightly with the empty or full condition of the neighbouring hollow viscera. Since they have an ovoid or almond shape they possess two surfaces, two ends, and two borders or margins; and the long axis is directed obliquely outwards and upwards towards the side of the pelvic brim. The anterior margin (hilum) is attached to the broad ligament, while the posterior is free; the surfaces are better described as outer and inner than anterior and posterior, and of these the latter is the more convex, the former being flattened; the lower and inner end (uterine pole) is the smaller, and is united to the uterus by the ovarian ligament; the outer end (tubal pole) is more rounded, and is attached to the fimbriated extremity of the Fallopian tube by the pen-shaped ovarian fimbria. Each ovary has dimensions which may roughly be expressed as length 3 cm., width 2 cm., and thickness 1 cm., or in inches $1\frac{1}{3}$ by $\frac{3}{4}$ by $\frac{3}{8}$; but the size varies much with (*a*) age, (*b*) menstruation, and (*c*) pregnancy, the maximum being attained about six weeks after parturition. The weight varies from 90 to 135 grains, falling as low as 20 grains in old age. The Fallopian tube lies above the ovary in the broad ligament, and its outer end encircles the outer end of the ovary; between the two and lying in the broad ligament is the parovarium. The ovary itself does not lie between the layers of the broad ligament, but is simply attached to the posterior surface of it by the hilum, at which point enter the vessels and nerves; here can be seen a white line (Farre-Waldeyer's line).

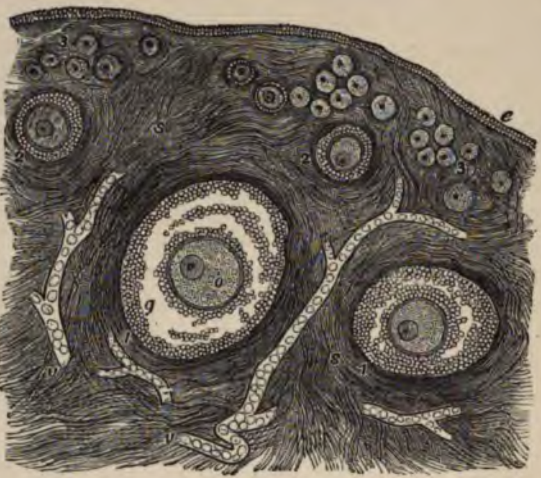

FIG. 5.—Section of ovary, showing large and small ovi-sacs, epithelial covering, and vessels. (After Baldy.)

The structure of the ovary is somewhat complicated. To the naked eye the gland has a pearly or even pinkish hue, with dusky blue spots here and there in it, which represent follicles containing ova, and it shows also yellow spots or old and ruptured follicles. In early life the surface is smooth, but later it becomes irregular. Two zones can be distinguished, a vascular zone or medulla or paroöphoron, and a parenchymatous zone or cortex or oöphoron. The former is softer in consistence, pink in colour, and contains large vessels, which enter from the broad ligament; the latter is firmer, white, and less vascular, and is dotted with small pits (follicles) (Fig. 5).

With the microscope it can be made out that the zona vasculosa is made up of loose connective tissue with some unstriped muscular fibres, large tortuous blood-vessels, nerves, and lymphatics, and some isolated tubular relics of the mesonephros. The parenchymatous zone shows most externally a layer of germinal columnar epithelial cells, which stops at the hilum at the white line, becoming continuous with the endothelium of the peritoneum of the broad ligament. Beneath this is in some ovaries (especially from old women) a connective tissue layer, the tunica albuginea. The greater part of the parenchymatous zone, however, is composed of the folliculiferous stroma. In it are found the Graafian follicles, from 36,000 to 40,000 in number in each ovary in the new-born infant, along with connective tissue, vessels, and nerves. The follicles are of three kinds, primary, growing, and ripe or mature: the primary and growing follicles are smaller than the ripe ones and lie nearer the surface, although occasionally a ripe one is found at the periphery. In the primary follicles are the primitive ova with a few somewhat flattened epithelial cells to form a covering; in the growing follicles the epithelial cells have become more cubical in shape, and form several layers round the ovum, which shows the commencement of a zona pellucida. The ripe follicle is a complicated structure: from without inwards can be recognised (*a*) a fibrous or external membrane or tunic, consisting of spindle-shaped, round, and stellate cells; (*b*) a membrana propria or internal tunic, consisting of a delicate layer of connective tissue; (*c*) a membrana granulosa or stratum granulosum, consisting of two or more layers of nucleated columnar cells, lining the follicle and massed together at one point into a clump of cells, forming the discus proligerus or cumulus oöphorus, and containing the ovum itself; (*d*) the liquor folliculi, a clear, yellow fluid, containing much paralbumin; and (*e*) the ovum, composed from without inwards of (1) the ovular epithelium, consisting of the granular epithelial cells of the membrana granulosa, which lie nearest to the ovum, and to which the name corona radiata is sometimes given; (2) the zona pellucida or vitelline membrane, which also shows a striation; (3) the perivitelline space; (4) the protoplasmic portion of the vitellus, consisting of an outer clear part and an inner more granular portion; (5) the deutoplasmic part of the vitellus; and (6) the germinal vesicle or nucleus, situated excentrically, and in it the germinal spot or nucleolus which has shown, according to some observers, amœboid movements. A ripe Graafian follicle, such as has been described, is ready to play its part in reproduction: in ovulation the follicle enlarges, passes from the centre towards the periphery of the ovary, during which time the ovum is throwing off two small rounded bodies, the polar globules, and the nucleus is undergoing karyokinetic changes; then the follicle ruptures, the ovum and part of its surrounding cells of the membrana granulosa escaping to be caught in the outer end of the Fallopian tube and carried along the tube into the uterus. The changes which occur in the follicle after rupture lead to the formation of the structure known as the corpus luteum. This structure is composed of blood-clot and of an ingrowth of the vascular wall of the follicle into the cavity from which the ovum has escaped; gradually the wall becomes thrown into folds and less room is available for the clot, which soon occupies only the centre of the corpus; the corrugated wall is first of a bright and later of a pale yellow colour; and finally the position of the corpus luteum is represented by a white cicatrix (corpus albicans). The corpus luteum which follows an ovulation unaccompanied by pregnancy runs through all the above-described stages much more quickly than one which ensues upon impregnation; otherwise there is no essential difference between the corpus luteum of pregnancy or corpus verum and the corpus luteum of menstruation (so-called). The colour is due to the presence of lutein.

The arterial supply of the ovaries is from the ovarians which come from the aorta; the veins pass to the pampiniform plexus in the broad ligaments and thence to the ovarian veins, of which one, the right, opens into the vena cava inferior and has a valve, while the other, the left, joins the renal vein at right angles, and has no valve. The lymphatics pass to the lumbar glands, and the nerves come from the inferior hypogastric plexus of the sympathetic and from the sacral nerves.

In the fœtus and young infant the ovaries lie above the plane of the brim of the pelvis, have an almost cylindrical elongated form, and show on section a much greater number of follicles than is to be observed in later life. The glands are chiefly derived from the germinal epithelium of the genital ridge; but a small part is developed from the mesonephros.

Fallopian Tubes.—The oviducts or Fallopian tubes are two sinuous tubes which occupy the upper part of the middle fold of the broad ligament, called the mesosalpinx or the mesentery of the tube. At the inner end they arise from the upper angle of the uterus, while the outer extremity opens into the general peritoneal cavity, but is connected with the ovary by the ovarian fimbria, and with the pelvic brim by means of the free fold of the broad ligament or infundibulo-pelvic ligament. Each tube is shaped like a shepherd's crook; the first part nearest to the uterus runs out straight, the second part curves outwards and forwards, and the third backwards and inwards. The first part, or isthmus, is the narrowest, and through it the uterine and tubal cavities are continuous; the second, or ampulla, is much wider; while the third, or infundibulum or fimbriated extremity, is expanded like the mouth of a trumpet and consists of the abdominal opening (ostium abdominale) and of the fimbriæ or fringes surrounding it. The right tube is usually a little longer than the left, the measurements being 11 cm. for the former and 10 cm. for the latter. The diameter near the inner end is about 4 mm., farther out it measures from 7 to 9 mm., and the ostium abdominale has a diameter of from 2 to 5 mm. A neck of constriction just internal to the infundibulum has been described; this may indicate an external sphincter of the tube. The fimbriæ are primary and secondary, or major and minor; the former are three or five in number; and in the intervals between them are the secondary, which are more numerous. The fimbriæ give to the end of the tube the appearance which has been termed "morsus diaboli" (Fig. 6).

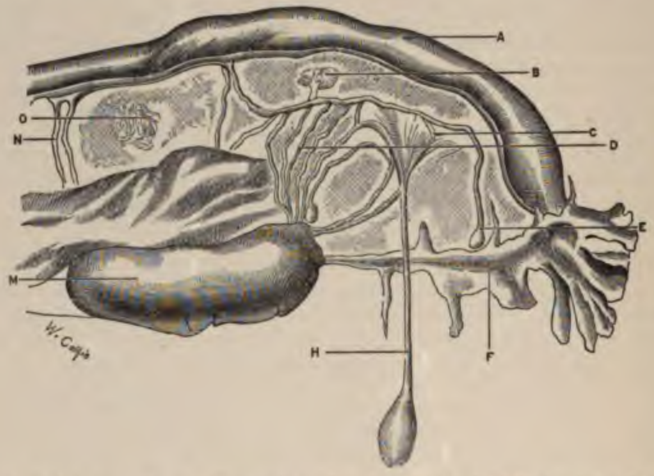

FIG. 6.—Fallopian tube, parovarium, and ovary (seen from the front). A, Fallopian tube; B, relic of mesonephros; C, curved tubules of parovarium; D, vertical tubules; E, cyst, of Gartner's duct; F, pen-shaped ovarian fimbria; H, pedunculated cyst of parovarium; M, left ovary; N and O, tubular relics of mesonephros. (After Ballantyne and Williams.)

The structure of the tubes is like that of other hollow viscera. To the naked eye three coats or layers are recognisable: an external or serous formed of the peritoneum of the mesosalpinx; a middle or muscular made up of an outer longitudinal layer of fibres and an inner circular; and an internal or mucous thrown into folds, which are continuous with the fimbriæ at the ostium abdominale. In the isthmus the tubal folds or rugæ are simple, and are arranged longitudinally, and the section of the tube is stellate; in the ampulla their arrangement is much more complicated, there are from three to five primary folds, and from eight to ten secondary ones, and all these branch, so that on section the tube has a dendritic appearance; and at the infundibular end the folds become continuous with the fimbriæ, primary folds with primary fimbriæ, and secondary with secondary. Under the microscope the outer coat has the ordinary appearance of a serous membrane; but it is noteworthy that at the ostium abdominale the flat cells of the peritoneum stop abruptly and give place to the ciliated columnar cells of the mucous membrane at the muco-peritoneal line, constituting a unique occurrence in the histology of the body. With regard to the muscular coat it is to be observed that there is a marked increase in the number of the circular fibres at the point where the tube passes through the uterine wall and also at the neck of constriction (above referred to) just internal to the infundibulum. The microscope has shown that the outer longitudinal and inner circular arrangement of the musculature is not constant, for in some specimens longitudinal fibres are found lying internal to the circular and bulging into the bases of the folds of the mucosa. A sub-mucous layer is not invariably present, but is occasionally found, especially in young women, as a thin layer most evident in the isthmus. It consists of round and spindle-shaped cells of embryonic character. The folds of the mucosa have a basis of similar cells with connective tissue fibres, a central artery, and are covered by columnar ciliated epithelium. The appearances of the tube in transverse section are distinctly suggestive of the presence of glands, but some authors deny their existence. To the outer end of the tube or to its fimbriæ there is sometimes found attached a stalked cyst; this is the true hydatid of Morgagni; it is not to be confounded with the pedunculated cysts growing from the parovarium. In the fœtus the tubes present several spiral convolutions.

Organ of Rosenmüller or Parovarium.—Between the folds of the mesosalpinx there lies, in addition to the Fallopian tube, the structure known as the organ of Rosenmüller or Parovarium. It consists of mesonephric tubular relics, and has also been termed the corpus pampiniforme or corpus conicum, or the epoöphoron. The organ is trapezoid, not triangular in form, and measures at its base from 1·5 to 3·5 cm. transversely, while at its narrow end near the ovary its breadth varies from 4 mm. to 3 cm.; its height is from 1·3 to 3 cm. It lies below the Fallopian tube and above the ovary, and is not free between the folds of the broad ligament, but is attached to the anterior one. It consists of a horizontal duct, the remnant of the upper third of the Wolffian duct, and of a number of tubules running more or less vertically downwards from it. The inner vertical tubules pass towards the hilum of the ovary, some reaching it, others falling short of it; the outer ones have a more curved course, and do not reach the ovary at all, but end in flask-like dilatations. The vertical tubules disappear near the hilum of the ovary in an obscure area (locus obscurior of Rosenmüller). In this place, as shown by the microscope, the tubules in an atrophied condition form a network, the rete ovarii, and some traverse the substance of the paroöphoron. The curved tubules most commonly lie in a free fold of the anterior layer of the mesosalpinx, and they often have attached to them cysts which have been called hydatids of Morgagni, but which really differ from that structure, as has been already stated. The horizontal duct shows a lumen, and has a wall composed of fibrous or connective tissue with some non-striped muscular fibres, and is lined with columnar non-ciliated epithelium. Few of the vertical tubules exhibit anything like a lumen; they have a thin covering of connective tissue, and are lined internally by columnar epithelium near their upper end, and by cubical cells nearer the ovary. Isolated tubular structures not included in the organ of Rosenmüller are sometimes found between that body and the uterus. These are also relics of the sexual part of the mesonephros, and probably correspond to the vas aberrans of Haller in the male. Granular areas may also be found at various spots between the layers of the mesosalpinx, some lying between the organ of Rosenmüller and the uterus, and others between the base of that organ and the Fallopian tube. In the fœtus and child they may be seen between the tubules of the parovarium itself. Sessile cysts sometimes take their origin in them. They consist of mixed tissue, partly fibro-cellular and partly fatty, with a trace of fine canaliculi with a choked lumen. Ballantyne and Williams think that they represent relics of the urinary part of the mesonephros, and that they correspond in the male to the organ of Giraldès or paradidymis of Waldeyer (Fig. 6).

UTERUS.—The uterus is a hollow, thick-walled organ lying in the middle of the pelvis between the rectum behind and the bladder in front. From it the menstrual discharge comes, and in it the phenomena of the development and growth of the fœtus take place. The question of its position, once much discussed, may now be regarded as settled. It is to a certain extent a movable organ, but its normal position, as determined both by clinical examination and by the study of frozen sections, may be defined as one of anteversion with a slight degree of anteflexion; it has a curve something like that of the anterior surface of the sacrum, and rests upon the posterior surface of the bladder; its lower end or cervix is directed downwards and backwards, and the whole organ is slightly twisted, so that its right upper angle is a little anterior to the left one. In shape it is a cone flattened from before backwards, with its base or fundus above, and its apex or cervix below. Below its middle third there is a slight constriction, the isthmus, which divides it into a body or corpus and a neck or cervix. The lower end of the cervix shows an opening, the os externum uteri; while the upper part of the body is the fundus, which is slightly arched, and to the angles of which are attached the Fallopian tubes. Its size varies with age, with certain physiological states, such as pregnancy, and of course with pathological conditions; but it may be stated generally that in the adult virgin it has a length of 7·5 cm. (3 inches), a breadth of 4 cm. (about 2 inches), and a thickness of 2·5 cm. (1 inch). In a woman who has borne children all these measurements are slightly increased. The length of the cavity from os externum to fundus is 6 cm. or 2½ inches. The thickness of the wall is 1 cm. Its weight is from 40 to 50 grammes. Approximately three-fifths of the whole length belong to the body of the uterus, and two-fifths to the cervix. The body has a form which is somewhat triangular in the virgin, and tends to the globular in a woman who has borne children; it has an anterior and a posterior surface, of which the former is flatter, two lateral borders to which the broad ligaments are attached, a superior border or fundus uteri which is free, two upper angles from which the inner ends of the tubes arise, and a lower end continuous with the cervix at the isthmus. A coronal section of the body shows the triangular shape of the cavity, while a sagittal section demonstrates that the anterior and posterior walls are in contact, the cavity being therefore a virtual one. The cervix is nearly cylindrical in form (it is slightly fusiform), and projects into the upper end of the vagina; its cavity is fusiform. It communicates with the cavity of the body at the isthmus by means of the os uteri internum, and with the vaginal canal by the os uteri externum. It has sometimes been divided into two parts, a supra-vaginal and a vaginal (portio vaginalis); but as it does not project equally into the upper end of the vagina, it is more correct to regard it as consisting of three portions, a supra-vaginal, an intermediate, and an intra-vaginal portion. The last named is the unattached part of the cervix; the intermediate is attached to the bladder in front, but is free behind; while the supra-vaginal is attached to the vaginal roof behind and to the bladder in front. The os uteri internum is a circular opening; the os uteri externum, or os tincæ, is a narrow, transversely situated orifice in the vaginal portion which divides this part of the cervix into two lips, anterior and posterior. It is noteworthy that the occurrence of labour shows that the uterus, which in the unimpregnated condition consists evidently of the two portions described above as body and cervix, really is made up of three parts, body, lower uterine segment, and cervix. Just before the opening up of the os uteri internum there can be recognised in the pregnant uterus the body or upper uterine segment with thick, contracted walls, the distended lower uterine segment, and the cervical canal. By some the lower segment is regarded as belonging to the body, and by others as due to the opening up of the upper part of the cervical canal. The former is the more probable view. The relations of the uterus to the pelvic floor will be considered later (Fig. 7).

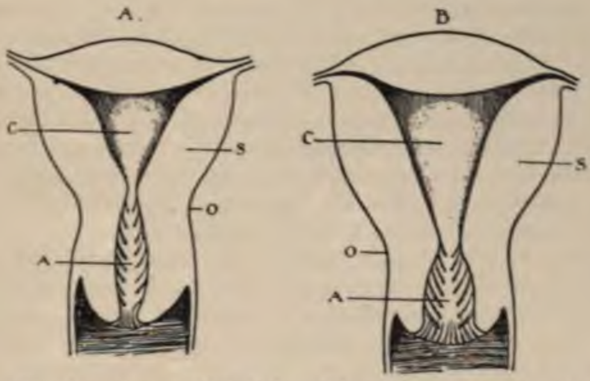

Fig. 7.—Coronal section of a nulliparous (A) and of a multiparous (B) uterus: A, cavity of the cervix and arbor vitæ; C, cavity of the body; O, constriction between body and cervix, the os uteri internum; S, wall of body. (After Tarnier.)

Structure of the Uterus.—With the naked eye it can be recognised that there are three coats or tunics—the external, serous coat, or perimetrium; the middle muscular coat, or mesometrium; and the internal mucous coat, or endometrium. The serous coat is formed by the general peritoneal lining of the pelvis. It may be noted that it covers the anterior surface of the uterus as low as the level of the os internum uteri, where it passes up on to the posterior surface of the bladder to form the utero-vesical pouch of peritoneum. Posteriorly it covers the posterior wall of the uterus, and descends as the pouch of Douglas behind the upper part of the vaginal roof as low as the level of the os externum uteri. From this point it ascends again on to the anterior rectal wall. Laterally the anterior and posterior layers of peritoneum meet at the borders of the uterus to form the broad ligaments of the organ. At the fundus the peritoneal coat passes directly from the anterior to the posterior surface. In the lower animals the musculature of the uterus is clearly divisible into two layers, an internal circular and an outer longitudinal, the former being a strongly developed stratum, and the latter a feebly marked one; but in the human uterus there is no such distinct arrangement. With the supervention of pregnancy, however, a notable hypertrophy of the muscular coat of the uterus occurs, and it is then possible to recognise three layers; but even then the distinction between them is never so marked as in the uteri of the lower animals. There is an inner layer immediately beneath the mucous membrane in which the fibres are arranged chiefly in longitudinal bundles. This has by some been regarded as a hypertrophied muscularis mucosæ. The middle layer is much the thickest, and while it is largely composed of fibres running in a circular direction, it also contains not a few which pass longitudinally, and from the peritoneal surface towards the mucous. In this way a thick feltwork is formed in the meshes of which lie the large blood-vessels of the uterine wall. The external layer is made up of longitudinal bundles with some obliquely and circularly disposed ones interspersed. Fibres from it pass into the broad ligaments, and also into the utero-sacral bands which run backwards to the sacrum below the peritoneal lining of the pouch of Douglas, dividing that pouch into three compartments, a central and a right and left lateral. The musculature of the cervix is largely continuous with the middle layer of the body. The mucous membrane is a thick coat, and has a smooth surface in the body of the organ, while in the cervical canal it is thrown into ridges, a vertical one with lateral ones slanting upwards and outwards from it, and to this arrangement the fanciful name of the arbor vitæ has been given. With the help of the microscope it can be made out that the serous coat has the ordinary histological features of the peritoneum, viz. a basis of delicate fibrous and connective tissue supporting large endothelial cells, while the muscular layer is found to be composed of very large unstriped muscle fibres, sometimes branched and with elongated nuclei, in a matrix of fine connective tissue and elastic fibres. In the muscular coat are also many arteries with thick walls and a convoluted intima, and large thin-walled veins with no valves. The microscopic characters of the mucous membrane of the body differ from those of the cervix, and require a separate description. Further, the appearances are much altered by such physiological conditions as menstruation and pregnancy. The following description refers to the mucous membrane of the resting (non-menstruating) virgin uterus :—

It measures 1 mm. in thickness. It is made up of a loose plexus of embryonic connective tissue cells branching and anastomosing with here and there more elongated and fusiform cells with oval nuclei; in the interstices are many leucocytes, and in its general appearance it suggests the structure of lymphoid organs. On the uterine surface is a single layer of ciliated columnar epithelial cells. On the surface also open the mouths of the uterine glands; these are tubular and extend down through the stroma, and often bifurcate at their lower ends, some of which reach and some even penetrate the muscular coat. Their direction is first perpendicular, and then oblique to the surface. They are lined by ciliated columnar epithelium upon a nucleated basement membrane, but the latter structure has not been recognised by all. Further, some observers regard these structures as not truly glandular in nature, but as simple pits or diverticula of epithelium, the mucous membrane being really a lymphatic tissue. In support of this view Leopold states that the connective tissue bundles are surrounded by endothelium, and therefore constitute lymph spaces. In the cervical canal the mucosa is thicker than in the body of the uterus; it is firmer in consistence, and its lining columnar epithelium is ciliated only on the tops of the ridges of the arbor vitæ. It possesses numerous racemose glands lined with cubical epithelium. At the level of the os externum uteri there is usually a sharp transition between the columnar epithelium of the cervical canal and the squamous epithelium reflected on to the outside of the vaginal portion of the cervix from the vaginal walls; but it is noteworthy that in the infant at birth there is often no marked line of separation between the two kinds of epithelium, the columnar lining of the cervical canal extending for a limited distance over the vaginal aspect of the cervix, and giving rise to the condition which has been called congenital cervical erosion. The so-called ovula Nabothi are retention cysts of the cervical glands.

During menstruation congestion of the mucous membrane of the body of the uterus occurs, accompanied by an exfoliation of a greater or smaller part of it; during pregnancy it gives attachment to the fertilised ovum, and forms the maternal part of the placenta.

The arterial blood-supply of the uterus is derived from two sources: the greater part comes from the uterine artery, a branch of the anterior division of the internal iliac, which passes in the broad ligament to a point near the os externum, where it turns upwards along the lateral border of the uterus, giving off transverse branches (the curling arteries) to that organ on the way, and one of these is large, is given off near the os internum, and uniting with that of the opposite side, forms the circular artery; a small part comes from the ovarian artery, whose terminal branches anastomose with those of the uterine. It is well to remember that the uterine artery which at a point opposite the os externum lies below and in front of the ureter then crosses over it and comes to lie behind its level. The veins of the uterus form a very rich network below the serous coat, and communicate with the vaginal and vesical veins, and also with the pampiniform plexus; they open into the internal iliac vein directly or into the ovarian vein. The lymphatics join those from the tube and ovary, and pass between the layers of the broad ligament to the lumbar glands; those from the cervix and upper part of the vagina pass together to the hypogastric and obturator glands. The nervous supply is derived in part from the hypogastric plexus of the sympathetic, and in part from the second, third, and fourth sacral spinal nerves; on each side of the uterus at the level of the isthmus these nerves unite in forming a large plexus sometimes called the cervical ganglion.

The uterus and vagina are developed from the fused portion of the two Müllerian ducts. The upper part of each duct remains separate as the Fallopian tube. In the new-born infant the uterus is partly an abdominal organ for a portion varying from a half to a third lies above the plane of the brim of the pelvis; the cervix is relatively much larger both in length and thickness than the body of the organ; and the folds of the arbor vitæ are prolonged in the interior quite to the fundus.

Ligaments of the Uterus.—The uterus partly is embedded in and partly rests upon the pelvic floor, but it is also to some extent supported by the various ligamentousl structures which sling it in the pelvic cavity. These are the round ligaments, the broad ligaments, the utero-sacral and the utero-vesical ligaments. The round ligaments are two fibro-muscular structures 13 cm. in length, which arise from the upper angles of the uterus; each passes in a curved direction to the internal inguinal opening, through the inguinal canal, and ends in the cellular tissue outside the external abdominal ring; and each is in its course enveloped in a fold (the anterior) of the broad ligament. They prevent downward and backward displacement of the uterus, a circumstance which explains the rationale of the Alexander-Adams operation of shortening these ligaments in cases of inveterate prolapsus uteri. The broad ligaments (ligamenta lata) are two double layers of peritoneum which pass outwards from the uterus to the lateral pelvic wall; perhaps it would be more correct to describe them as the mesentery of the uterus, for that organ really lies between the folds of peritoneum of which the ligaments are composed; at any rate a part of each ligamentum latum forms a mesentery for the Fallopian tube (mesosalpinx). The base of the ligament may be represented as a wavy line passing outwards to the lateral pelvic wall at a point just in front of the sacro-iliac synchrondrosis; the upper (free) margin passes from the upper angle of the uterus outwards to a point in the ilio-pectineal line midway between the sacro-iliac joint and the pectineal eminence; the inner attachment is along the lateral border of the uterus from the upper angle almost to the lateral part of the roof of the vagina; and the outer edge is attached to the pelvic wall as low down as the level of the ischial spine. Three folds of the broad ligament have been recognised: an anterior, in which lies the round ligament (ligamentum teres); a middle, the mesosalpinx, in which are situated the Fallopian tube and the parovarium and vessels and nerves; and a posterior, to which is attached the anterior margin of the ovary. The utero-sacral ligaments are two muscular bands which pass backwards from the upper part of the cervix uteri to the sacrum, and enclose the rectum on the way; in the anterior part of their extent they raise up the peritoneum of the pouch of Douglas into two folds, the recto-uterine ligaments, which serve to subdivide the pouch into compartments. The utero-vesical ligaments are simply peritoneal folds which pass from the cervix to the posterior surface of the bladder and mark off a slight depression, the utero-vesical pouch. The ovarian ligament and the infundibulo-pelvic have already been described.

INTERMEDIATE ORGAN: THE VAGINA.—By means of the musculo-membranous canal, the vagina, the internal genital organs are put in communication with the exterior; it may therefore be termed the intermediate organ. It forms a slit in the pelvic floor, lined with mucous membrane, extending from the hymen to the cervix uteri, and lying between the urethra and bladder in front, and the rectum and perineum behind. With the woman in the erect posture the vagina makes an angle of about 60° with the horizon, and is therefore nearly parallel to the plane of the pelvic brim. When the bladder is empty the normal axis of the vagina forms with the long axis of the uterus a right angle; but when the bladder is distended the angle becomes more or less obtuse. When the rectum is full the vaginal axis becomes almost perpendicular. Normally the vaginal walls are in contact, and a sagittal section of the pelvis shows it as a linear slit, while a transverse section reveals an H-shape; it is correct to describe the vagina as a tube only when the finger or instruments are being passed up it, or when the fœtus is being propelled down it. A cast of the distended vagina has the form of a truncated cone, base uppermost. It has two walls, a lower or vulvar end, and an upper end, roof, or fornix. The anterior wall is the shorter, measuring 5 cm.; the posterior has a length of 7·5 cm. The anterior wall has a somewhat triangular shape with the base above and the apex at the vaginal orifice; and its surface is thrown into numerous folds, there being one median longitudinal ridge, the anterior column, and many transverse and oblique rugæ running off from it; these rugæ are continued on the inner aspect of the hymen; and they disappear to a large extent after parturition. On the posterior wall the rugæ show a similar but less marked arrangement; this wall also has a triangular shape, but instead of being straight, as is the anterior, it has a sigmoid curve; like the anterior wall it is reflected upon the cervix forming part of the vaginal roof or fornix. The lower end of the vagina opens at the vulva between the labia, situated laterally, the hymen behind, and the vestibule in front; it is an antero-posterior slit. The roof of the vagina through the projection of the cervix uteri into it is divided in imagination into four parts, called the anterior, posterior, and lateral fornices or fossæ. These fornices, of course, only exist when the vagina is distended; the posterior is much the deepest. The relations of the vagina to surrounding parts are of considerable importance. Anteriorly it is connected in its upper half with the bas-fond of the bladder, and with its neck by dense areolar tissue which forms the vesico-vaginal septum; in its lower half it is intimately united to the urethra, which is indeed embedded in it, the two together forming the urethro-vaginal septum. Posteriorly the vagina in its upper one-fourth is separated from the rectum by the pouch of Douglas; in its middle two-fourths it is connected by loose connective tissue with the rectum; while in its lower fourth the perineal body lies between it and the rectum and anus. It is important to remember the relation of the posterior fornix to the peritoneal cavity, and it is noteworthy that sometimes the pouch of Douglas is deeper than normal, and may descend between the vagina and rectum almost to the perineum. Laterally the vagina is related to the levatores ani muscles, and to the large venous plexuses. The lateral fornices are related to the bases of the broad ligaments and to the vessels lying in them. The anterior fornix is distant a little over an inch from the bottom of the vesico-uterine pouch of peritoneum; to its outer sides and well above it are the ureters (Fig. 8).

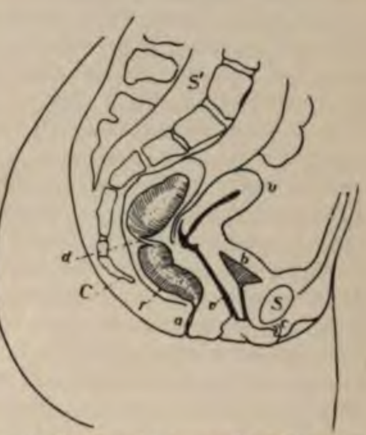

FIG. 8.—Diagram of a supposed mesial section of the pelvis of a living woman: *a*, anal canal; *r*, rectum; *v*, vagina; *c*, clitoris; *b*, bladder when collapsed; *u*, uterus; *d*, valve of rectum (Houston); *S*, symphysis pubis; *S'*, sacrum; *C*, coccyx. (After Dorland.)

The structure of the vagina as seen by the naked eye consists of an outer layer or coat of connective tissue and elastic fibres, by means of which it is united to surrounding structures, and in which lie large venous plexuses; of a middle or muscular coat in which separate layers of fibres have been described, concerning the arrangement of which, however, there is a want of unanimity among anatomists; and of an inner layer or mucous membrane, thrown into folds and situated upon a submucosa, of a pink colour usually, but becoming purple in pregnancy. Microscopically the muscular coat is found to consist of unstriped fibres running in bundles in various directions, and between which lie large veins and lymphatics. The submucosa consists of loose areolar tissue which enters into the formation of the columns, and on this lie several layers of pavement epithelium; there are many papillæ and only a few true secreting glands. The mucous membrane, if exposed continuously to the influence of the air, as in prolapsus, becomes skin-like, and it is in many respects a cutaneous rather than a mucous structure. If it be true that it is developmentally derived from the lower ends of the Wolffian ducts (Wolffian bulbs of Hart) which are epiblastic structures, this peculiarity is largely accounted for. The vagina itself is developed from the Müllerian ducts (fused), but it is quite possible that its lining membrane is, as Hart maintains, a derivative of the Wolffian bulbs. In the fœtus the vagina is often distended with much desquamated epithelium, and in vertical mesial section looks as if it were almost the only pelvic content. Great discussion has recently taken place as to the micro-organisms of the vagina. The consensus of opinion now seems to be that normally no pyogenic cocci are present, although many bacteria are to be found, including the thick bacillus vaginalis of Döderlein. The arterial blood-supply of the vagina is from the vaginal arteries (from the internal iliacs), from branches of the uterine arteries, and from branches from the pudendal arteries. An azygos branch in the anterior wall joining the circular artery of the cervix has been described by Hyrtl. The veins, without valves, form complete sheaths for the canal, both outside the muscular coat and beneath the mucous membrane. The lymphatics from the lower fourth of the vagina join those from the external genitals, and pass to the inguinal glands; those from the rest of the canal go to the internal iliac glands. The nerves are from the inferior hypogastric plexus of the sympathetic, from the fourth sacral and from the pudic nerves.

External Generative Organs.—The external genitals collectively constitute the vulva or pudenda, and consist of the labia majora and minora situated laterally, and of the mons veneris, clitoris, vestibule, meatus urinarius, vaginal orifice, hymen, fossa navicularis, fourchette, and perineum in the middle line. The *mons veneris* is an irregularly triangular area of skin with underlying adipose and connective tissue situated in front of the symphysis pubis. It is covered from the time of puberty onwards with crisp, curly hair which rarely passes upwards towards the umbilicus, and in this respect therefore differs from the hair of the same region in the male. Numerous sebaceous and sudoriparous glands are found in the skin of the mons, and in its substance also the external ends of the round ligaments terminate in the form of scattered muscular fibres. From the sides of the mons two folds of skin pass backwards towards the perineum, forming in their course the lateral boundaries of the vulva; these are known as the *labia majora*, or *externa*, or alæ majores. Each labium has on cross section a somewhat triangular form, the base rests on the pubic ramus, and the sides are the external and internal surfaces. The external surface is covered with rugæ, and has an appearance recalling that of the scrotum in the male; it carries numerous hairs from the age of puberty onwards, and there are plentiful sebaceous glands. On the inner surface the hairs are scanty, and the colour is less dark than that of the outer aspect. In multiparous women and in the old or emaciated the labia are pendulous, and the vulvar aperture gapes, but in nulliparæ and in young or stout persons they are in contact and conceal the underlying structures. The labia are covered externally with a layer of skin, with numerous hair follicles and sebaceous glands; below this are fibres of unstriped muscle resembling those of the dartos in the male, and beneath them is a cellulo-adipose layer lying upon an elastic fibrous sac (Broca's sac). Sweat glands are present on the outer aspect of the labia. The labial lymphatics pass to the superficial inguinal glands; the arteries are branches of the external and internal pudic and of the epigastric; the veins go to join those of the bulbs and the vaginal veins; and the nerves are from the internal pudic and small sciatic. Posteriorly the labia fuse insensibly with the skin of the perineum (Fig. 9).

The *labia minora* or *interna*, or the *nymphæ*, are two slender folds of skin resembling mucous membrane which lie inside the labia majora, and form the lateral boundaries of the vestibule and of the vaginal aperture. They are directed backwards and outwards, diverging from the middle line. Each labium has a smooth outer surface apposed to the inner surface of the labium majus, but separated from it by a deep groove; an internal surface usually in apposition with the internal aspect of the labium of the opposite side, and showing at its base a white line; a free margin, irregular and fringed; an attached margin arising from the side of the vestibule anteriorly, and from the inner aspect of the labium majus of the same side posteriorly; a posterior end which, according to some anatomists, blends with the labium majus about its middle, and according to others is joined with the corresponding part on the opposite side to form a thin fold lying in front of the perineum and called the *fourchette;* and an anterior end which bifurcates at the level of the clitoris, and one part goes above the clitoris to unite with that of the opposite side to form the prepuce, while the other passes below the clitoris to meet that of the opposite side in the frenulum. The nymphæ contain no adipose tissue and do not carry hairs, otherwise they have the structure of skin. The sebaceous glands open alone on the surface. The nymphæ have the same vascular and nervous supply as the labia majora. The *clitoris* is a small curved body with a prepuce and frenulum formed from the anterior ends of the labia minora in the way that has been described above. It lies in the middle line at the apex of the vestibule, and is the homologue of part of the male penis. It consists of the glans, a mass of erectile tissue not larger than a pea and covered with fine skin, and of the body or corpus clitoridis, a cord-like structure which passes to the anterior edge of the pubic arch, where it divides into two crura, the inner margin of each of which is covered by the erector clitoridis muscle. The nerves of the clitoris are very numerous, and end in end-bulbs, and also in the peculiar genital corpuscles of Krause; they come from the pudic. The arterial supply consists of the terminal branches of the internal pudic, a dorsal twig to the glans and prepuce and profunda branches to the corpora cavernosa. There is a dorsal vein which ends in the vesical plexus; the lymphatics pass to the inguinal glands.

The *vestibule* is a triangular area with at its apex the clitoris (just described), with its sides formed by the inner margins of the labia minora, and having for its base the upper margin of the vaginal orifice. At the middle of the base is the meatus urinarius, and surrounding the meatus and stretching up from it towards the clitoris is a band known as the male vestibular band. This band is not always seen in the adult, but is fairly evident in the new-born infant. Under the mucous membrane of the vestibule some veins are found running transversely from one side to the other: these form the pars intermedia, and join together the two vestibular or vaginal bulbs which are placed at the sides of the vestibule. The surface is covered by several layers of pavement epithelium, below which are papillæ with capillary loops, and beneath them a connective tissue layer; there are compound racemose glands. The *meatus urinarius* is the external orifice of the urethra, and is a small dimple in the mucous membrane of the vestibule. The urethral canal at this point is a small vertical slit; immediately within it are the openings of two short tubes (Skene's tubules) which lie in the muscular wall of the urethra, and which are said by some to be relics of Gärtner's ducts derived from the lower ends of the Wolffian ducts, and by others to represent the male prostate. Round the meatus are little openings, the orifices of the mucous glands of the vestibule (glandulæ vestibulares minores) already referred to. The meatus lies from 2 to 2·5 cm. below the clitoris.

The *orifice of the vagina* lies immediately behind the vestibule and in front of the fourchette, which it will be remembered was formed by the posterior union of the labia minora. It is an antero-posterior slit, and in the virginal condition is partly occluded by the hymeneal membrane. Between the hymen and the fourchette is a small depression to which the fanciful name of fossa navicularis has been given. The *hymen* has usually a crescentic or semilunar shape, but occasionally it is a diaphragm with a single central aperture, or with two apertures, or rarely with several openings in it. It presents a free border towards the vaginal orifice which is thin and concave, an attached border posteriorly, an inferior or external surface, and a superior surface on which the vaginal rugæ can sometimes be traced. It is composed of connective tissue with arteries and veins in it and many elastic fibres. It is usually but not invariably torn at the time of the first coitus, and it is certainly broken up when the first child passes through the vaginal orifice. Portions of the hymen remain as little fleshy projections (three to six in number), which are known as the carunculæ myrtiformes. Behind the hymen are the *fossa navicularis* and the *fourchette*, which have been already described; it may be noted that the fourchette is very often torn in the first parturition. Behind the fourchette is the skin covering the base of the perineal body. Reference in passing has been made to the *vaginal* or *vestibular bulbs*. These are elongated masses of veins lying at the sides of the vestibule and vaginal orifice in close relation with the constrictores vaginæ muscles. They are about the size of a bean, but in an injected condition have a length of about 4 cm.; they have a somewhat conical form, and their apices lie at the level of the meatus urinarius, where they communicate by the pars intermedia, while their bases are situated opposite the lower third of the vaginal orifice; and they consist of complicated venous plexuses in a fibrous sheath. They communicate freely with surrounding veins. In addition to the sudoriparous, sebaceous, and mucous glands of the vulva already referred to there remain for description two structures known as the *vulvo-vaginal glands*, glandulæ vestibulares majores, or the glands of Bartholin, Duverney, or Méry. These are two small oval bodies, having the size of a bean or an almond, and situated on either side of the vaginal orifice near the posterior ends of the vaginal bulbs. They lie beneath the superficial perineal fascia, are racemose, mucous, or sero-mucous glands, consist of numerous acini lined with columnar epithelium, and discharge their secretion by an excretory canal which opens at the point of union of the posterior third with the anterior two-thirds of the vaginal orifice in the furrow separating the hymen from the internal surface of the labium minus.

The clitoris is the representative of the genital tubercle of the embryo, and the labia are the edges of the genital cleft. Behind the genital tubercle there is in the early stages of development a cloaca common to the urogenital canal and to the rectum; but through the growth of a septum which becomes the perineal body the rectal canal is separated from the space in front known as the urogenital sinus. Later the vesico-vaginal septum divides the urogenital sinus into two parts, the urethra and the vagina. According to some, the hymen is simply the lower end of the vaginal mucous membrane everted; according to others, it is formed from the external genitals; and according to Hart, it is due to the breaking down of the Wolffian bulbs from which the vagina receives its epiblastic lining.

THE MUSCULAR AND CONNECTIVE TISSUES lining and bridging across the pelvic cavity now fall to be described. The pelvic peritoneum with its pouches and duplicatures has been considered, but it is necessary to refer to the muscular structures which lie below it and modify by their presence the diameters of the osseous pelvis. The iliacus muscle on each side lessens the depth of the internal iliac fossa; while the psoas muscle and the vessels near it change the shape of the pelvic brim and diminish the transverse diameter of the brim by about 1 cm. The iliacus and psoas muscles together increase the depth of the pelvic cavity. It may also be noted here that the presence of the rectum causes the left oblique diameter at the brim to be a little less than the right. The obturator internus and pyriformis muscles narrow the transverse and the oblique diameters of the cavity, while the bladder, urethra, and rectum diminish its antero-posterior diameter. The most important soft parts of the pelvis, however, are those which fill up the pelvic outlet and form the pelvic floor or diaphragm; to the description of these some space must now be given.

The PELVIC FLOOR is composed of skin, mucous membrane, fasciæ, muscles, and fat; into its composition it may be counted also that the bladder and rectum enter; and although it forms a compact diaphragm it is nevertheless a diaphragm traversed by clefts or lines of displacement, represented by the vaginal and urethral canals. The floor may be studied first by dissection, and second by sections; in this way its dissectional and its structural anatomy are both brought out. On the inner aspect of the pelvic floor lie the uterus, the Fallopian tubes, broad ligaments, ovaries, and the pelvic peritoneum; and on its outer aspect lie the external organs of generation; all these structures have been described. The dissection of the floor is usually undertaken from without inwards. Its skin surface shows a median depression consisting of an anterior cleft in which lie the external organs of generation and a posterior or natal cleft in which is situated the anal aperture. The anus is about 1 inch in length, and is parallel to the axis of the brim of the pelvis, and is in consequence at right angles to the axes of the vagina, rectum, and urethra, for these are all parallel to the plane of the brim. It lies about 4 cm. behind the hymen or fourchette, being separated therefrom by the skin over the perineal body. It has an internal and an external sphincter. The part of the pelvic floor which projects beyond the plane of the outlet of the osseous pelvis is termed the *pelvic floor projection;* it measures about 3 cm. Below the skin of the posterior part of the pelvic floor are situated the superficial fascia, and on each side the base of the ischio-rectal fossa. This fossa has an outer wall formed by the obturator internus, an inner and upper wall composed of the levator ani and the anal sphincter; its apex is the point of junction of these two muscles, and its base lies between the transversus perinei and the edge of the gluteus maximus. It contains much fat. Under the skin of the anterior part of the floor lies the superficial fascia with its deep layer attached to the pubic arch, and passing round the transversus perinei to join the anterior layer of the triangular ligament. When the fascia is dissected off the perineal muscles three pairs are revealed. They form on each side an isosceles triangle with base posterior; the base is represented by the transversus perinei arising from the ramus of the ischium and inserted into the perineal body; the outer side is formed by the erector clitoridis which passes from the ischial tuberosity to the crus clitoridis; and the inner side is constituted by the bulbo-cavernosus or constrictor vaginæ which arises in the centrum tendineum of the perineal body, and along with the corresponding muscle of the opposite side forms a sort of sphincter for the vaginal orifice. Below the posterior end of the bulbo-cavernosus lies the gland of Bartholin, and farther forwards and also below the muscle is the vaginal bulb. Another muscle which finds an attachment in the perineal body is the external sphincter of the anus. In this place a few words of description regarding the perineal body are necessary. It is found on dissection to be a somewhat pyramidal structure filling up the space between the anus and the vagina, which is produced by the fact that these two canals diverge from one another, the vaginal axis passing forwards and the anal backwards. It is composed of muscular origins and insertions and of fibrous and elastic tissue. The muscles passing into it are the sphincter ani, the two transversus perinei muscles, the two bulbo-cavernosi, and the levatores ani. Its base is covered by the skin between the anus and the vaginal orifice, and constitutes the part torn in perineal lacerations, its anterior side is related to the posterior wall of the vagina, its posterior side to the anterior rectal wall and anus, while laterally there is adipose tissue. Its vertical measurement is about 3·5 cm., and its antero-posterior about 2·5 cm. The structural integrity of this body is an important factor in the solidity of the pelvic floor, for when it is seriously damaged prolapse of the vaginal walls and uterus takes place. If the muscles which have been described be now removed the anterior layer of the triangular ligament is exposed; behind it are the pudic vessels and nerves and the posterior layer of the triangular ligament. Neither the perineal muscles, however, nor the triangular ligament constitute the most powerful supporting elements of the pelvic floor; these are supplied by the levatores ani and the coccygei muscles. The levatores ani have the form of a horse-shoe open in front, and sling the vagina and rectum across the pelvic cavity; they arise from the back of the pubic bone, from the pelvic fascia along the white line, and from the ischial spine, and they are inserted into the rectum, vagina, tip of the coccyx, and into each other. Each coccygeus muscle arises from the spine of the ischium and the inner surface of the pelvic fascia, and is inserted into the coccyx and the lower part of the sacrum; it supplements the levator ani. The bladder, rectum, retro-pubic fat, along with blood-vessels and nerves and the pelvic fascia, make up the rest of the pelvic floor. The pelvic fascia is a somewhat complicated structure. It is continuous above with the iliac fascia, and at a point a little below the level of the brim, called the white line, it divides into a layer which covers the obturator internus (obturator fascia), into a thin sheet which lies on the under surface of the levator ani muscle (anal or ischio-rectal fascia), and into the great visceral or recto-vesical layer which covers the upper surface of the levator ani and passes to the bladder, vagina, and rectum, where it divides into four layers—vesical, vesico-vaginal, recto-vaginal, and rectal. The layers of the triangular ligament to which reference has already been made are connected with the obturator and recto-vesical divisions of the pelvic fascia.

The remaining component parts of the pelvic floor and the relation of the parts of the floor to each other are well brought out by the sectional method of study. A vertical mesial section of the pelvis and its contents shows that the pelvic floor is made up of two segments divided by the slit of the vagina which runs parallel to the plane of the brim (Fig. 8). Hart has given to these segments the names of pubic and sacral. The pubic or anterior segment is composed of the urethro-vaginal septum and the urethra, of the bladder, and of the pad of fat lying between the bladder and the posterior surface of the symphysis pubis, and called the retro-pubic fat; it is somewhat triangular in form; it is loosely attached to the pubic bones; and it is bounded posteriorly by the vagina and in front by the symphysis. It may be called the displaceable segment of the floor. The posterior or sacral segment is composed of the posterior vaginal wall, of the perineal body, of the muscles and connective and fascial structures surrounding the rectum and anus, and of the rectum itself; it is roughly quadrangular in form; is firmly attached to and dovetailed into the sacrum and coccyx, and it is bounded in front by the vaginal slit, and behind by the posterior pelvic wall. It may be called the fixed segment of the pelvic floor. In the resting condition of the floor the two segments are in close contact along the line of the vaginal cleft, for intra-abdominal pressure presses the pubic against the fixed sacral segment, and so produces a supporting floor on which rests the uterus and annexa and the intestines. It has sometimes been stated that the uterus should be regarded as a part of the floor, but it is undoubtedly more correct to regard it as a viscus resting on the anterior segment of the floor. Under certain conditions the segments can be separated. Thus in prolapsus of the uterus the floor is weakened by general laxity of tissue, or more often by actual destruction of the supporting sacral segment, with the result that the anterior segment with the superposed uterus passes downwards past the sacral segment and protrudes from the vulva. In labour, on the other hand, the anterior segment is pulled up into the abdomen, the posterior is pushed backwards, and in the space between the fœtus descends on its way out of the abdomen. Again, if the patient be placed in the genu-pectoral posture, and the vaginal orifice kept open (as by a Sims speculum), the vaginal cleft becomes a cavity, and the cervix uteri is seen to be at a greater distance from the vulva; in this case also the segments of the floor have separated, the anterior passing towards the diaphragm; and this peculiarity has been utilised in the examination of the pelvic viscera and in the treatment of morbid conditions of the uterus and vagina. Yet again, when the volsella is attached to the cervix uteri and downward traction made in the axis of the vaginal cleft, the uterus can be drawn to the vulva without the use of force and examined or operated on; here the anterior segment of the floor has been pulled down past the sacral.

The Bladder and Rectum.—In order to complete this survey of the female genital organs and of the structures in immediate connection with them, it is necessary that the *bladder* and *rectum* be described. The bladder when empty usually forms with the urethra a Y, but occasionally its cavity forms a slit continuous with the urethra, and the mucous membrane is thrown into folds; the former is the diastolic and the latter the systolic empty bladder. Further, in the adult woman it is a pelvic content when empty, but in the infant and fœtus it normally lies above the plane of the brim. The upper part alone is covered with peritoneum in the adult. Its wall is composed of several layers of unstriped muscle, and internal to these is a mucous membrane consisting of connective tissue lined by several layers of transitional or multiform epithelium. The mucous and muscular coats are separated by a loose submucosa. There are three openings into the bladder, the internal orifice of the urethra and the orifices of the two ureters. It is necessary, in connection with the surgery of the genital organs, to remember the course of the ureters in the pelvic cavity; each ureter having crossed the iliac vessels passes downwards, backwards, and a little outwards on the wall of the pelvis to a point near the ischial spine where it bends downwards, forwards, and inwards, and passes beneath the base of the broad ligament, and is crossed by the uterine artery about the level of the os uteri internum; it then passes at the side of the upper third of the vagina, runs for a distance of about 1·5 cm. in the vesico-vaginal septum, and then traverses the bladder wall in a direction obliquely downwards and inwards to open at one angle of the trigone. The urethra is a straight slit measuring about 4 cm. in length. It is parallel to the vagina, and is indeed embedded in its anterior wall. It is lined with a mucous membrane covered with squamous epithelium in its lower part, and with transitional epithelium in its upper part; it has also a muscular coat and a submucosa. Its external orifice with Skene's tubules has been already described.

The rectum extends from the left sacro-iliac joint to the anus, and is divided into three portions. The first has a mesentery (meso-rectum) and ends at the third sacral vertebra; the second part shows reflection of the peritoneum on to the upper part of the vaginal wall; and the third has no relation to peritoneum at all, and lies behind the posterior vaginal wall, separated from it by loose tissue and lower down by the perineal body. It is lined with a mucous membrane covered with columnar epithelium and showing numerous Lieberkuhnian follicles. There are oblique folds consisting of the mucous membrane of the submucosa, and of some circular muscular fibres; one of these lies near the level of the sacrum, another about 4 cm. above the anus, and a third intermediate. The lowest of the three is called the sphincter tertius or valve of Houston (Fig. 8). In the lower part of its extent the rectum runs parallel to the vagina, but the anal canal turns sharply backwards and is at right angles to the vaginal axis.

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Arrested Developments of

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See also HERMAPHRODITISM.

The *malformations of the female genital organs* are mostly instances of arrested developments, that is to say, in them we see conditions which are temporary in normal organogenesis and which have become permanent. For instance, the uterus which is originally composed of two lateral halves (the Müllerian ducts) is normally an organ which shows no trace of its temporary duplicity, but which may, under certain circumstances, exhibit signs of it, as is evident in the various types of "double uterus." In some few cases this comparatively simple explanation of genital malformations fails, as in the rare anomaly known as trifid uterus and in double vulva; but, speaking generally, a malformation of one or other of the genital organs represents a state of affairs which at an early stage of development would be normal.

Malformations of the ovaries have an important bearing upon the fertility and health of the individual who is the subject thereof, for the ovary is not only the gland which forms the ova, but it is also in all probability a structure which secretes a product without which perfect health is impossible. *Complete absence* of both ovaries is rare except in monstrosities, even *unilateral absence* is uncommon; but a rudimentary state of the glands is more frequent. *Rudimentary ovaries* show only a small number of ill-developed ovisacs in a considerable quantity of connective tissue; and they are not uncommonly displaced into the inguinal canals, or lie at a higher level than is normal. The clinical importance of these defective conditions depends greatly upon their unilateral or bilateral character; in the former case reproductive activity may be little interfered with; in the latter the secondary sexual characters will be absent, the menstrual function will not be established, and sterility will be inevitable. There may also be associated morbid states, such as epilepsy, chlorosis, and the like. The diagnosis is often impossible without a laparotomy, although there may be a strong presumption in favour of defective ovaries. It has to be borne in mind that the uterus may be normal. *Accessory* or, more correctly, *constricted ovaries* are to be found in from two to three per cent of autopsies: they are about as large as a pea, and are generally attached to the normal ovary by a pedicle; they may contain Graafian follicles, and are due either to fœtal peritonitis causing separation of a piece of the ovary or to budding of the primitive sexual gland; and their presence probably accounts for the persistence of menstruation or the occurrence of pregnancy after a double ovariotomy or oöphorectomy. *Congenital hernia of the ovary* is nearly always inguinal, for, through the persistence of the canal of Nuck, the gland passes down into the inguinal canal and may reach the labium majus; in such cases the swelling in the groin is subject to periodic monthly enlargement, a circumstance which makes the diagnosis of its nature possible. It may give rise to much pain and to reflex phenomena, and must then be excised. It is often associated with the uterus unicornis and hernia of the Fallopian tube, as has recently been brought out by Browne (*Trans. Amer. Gyn. Soc.* xxiii. 352, 1898).

The *malformations of the Fallopian tubes* can hardly be diagnosed during life save by the opening of the abdomen. Both tubes may be *absent*, but more commonly *one alone is wanting*, and then it is frequently found that there is also a uterus unicornis and unilateral absence of the ovary. The tubes also may be present in a *rudimentary* or *solid condition*, an anomaly which is practically equivalent to their absence. The *spiral convolutions* which normally exist in antenatal life may persist as abnormal twists in adult life; possibly they may be the causes of dysmenorrhœa and sterility. *Accessory tubal ostia* and tufts of fimbriæ are not uncommon (three to six per cent); as many as six have been seen in one case; they are usually situated near the outer end of the tube; and it has been thought that they may sometimes be the cause of extra-uterine gestation.

The *malformations of the uterus* may be divided into those showing apparent excess in formation, those due to defect, and those due to displacement. With the exception of the extremely rare *trifid* or *accessory uterus*, the various forms of *double uterus* are only apparently due to excessive formation; they are really defective developments, being caused by more or less complete want of fusion of the portions of the two Müllerian ducts which normally unite to make the single uterus. The most complete want of fusion is seen in the *uterus didelphys, s. duplex*. In it there appear to be two single uteri lying side by side, but each possessing only one ovary and tube; there may also be two vaginal canals and a double opening in the hymen. The *uterus bicornis* is a much commoner type; in it the upper part of the uterine body is evidently separated into two horns or halves, while the lower part of the body and the cervix are single; in its most marked form the two horns are wide apart, and between them passes a band (recto-vesical) from the bladder to the rectum; but in the slightest degree there is only a notch or groove at the fundus to indicate externally the double nature of the organ (*uterus cordiformis*). One or both horns may be imperforate, and the cervical canal may be double or single. In its external appearances the *uterus septus* or *bilocularis* gives no indication of its double character, yet it is divided more or less completely into two cavities by an internal septum running usually antero-posteriorly. The septum may be present in the whole extent of the organ from fundus to external os, or it may be absent above and present below, or *vice versa*, or yet again the septum may be incomplete in the middle. There may also be a certain degree of rotation of the uterus so that the cavities come to lie antero-posteriorly. All the forms of double uterus are difficult of diagnosis, the conditions with which they are most apt to be confounded being fibroid tumours and extra-uterine pregnancy. The occurrence of a regular menstrual discharge every fortnight will excite suspicion, and then the physical examination may reveal a double cervical orifice, or the sound may pass into a double cavity; but many times the malformation has not been discovered till labour has come on and called for manual interference, for the double uterus does not entail sterility, although it may predispose to malpresentations. Pregnancy may occur in one-half of the double uterus and menstruation may continue from the other, or decidual membranes may form in it; pregnancy may also occur in both horns simultaneously, or at different but not widely separate dates; or again the two horns may become impregnated alternately. When the half in which the fœtus lies is rudimentary in its development the case is clinically one of extra-uterine gestation.

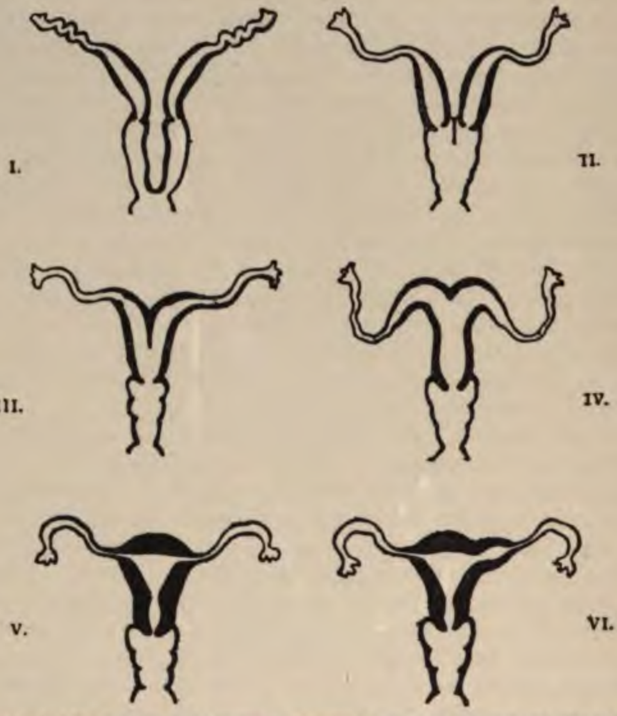

FIG. 10.—Diagrams of the tubes, uterus and vagina in different mammals, representing also the malformations which occur in man. I. Double uterus and vagina (some marsupials); II. double uterus, vagina partly divided by septum (rabbit); III. uterus single below but double above (most rodents and cheiroptera); IV. uterus single but with two horns (uterus bicornis of ungulata and carnivora); V. single uterus (man and apes); VI. uterus with rudimentary cornu (uterus unicornis cum cornu rudimentario of man). The uterus unicornis of man is not figured. (Fothergill.)

The *uterus unicornis* is due to the absence of one Müllerian duct; sometimes, however, the name is also given to the anomaly in which the second horn is not altogether absent, but exists as a solid or hollow rudiment, representing an imperfect development of one Müllerian duct. The uterus unicornis inclines to one side, and tapers to a point which is continuous with the Fallopian tube, and is marked by the attachment of the round ligament. The cervix and vagina are usually small and poorly developed. The diagnosis is rarely made during life, for menstruation and even pregnancy may occur normally; but pregnancy in the rudimentary horn attached to the uterus unicornis is a serious circumstance, having all the prognostic significance of ectopic gestation, rupture usually taking place into the peritoneal cavity.

The term *uterus rudimentarius* has no very precise pathological signification, for it has been applied to the cases in which the organ was reduced to a strip of muscular tissue lying between the layers of the broad ligament (*uterus membraniform* or *membranaceus*), to those in which there was a fair-sized fibro-muscular uterus, but without a cavity or with a very incomplete one (*uterus solidus, uterus partim excavatus*), and to those in which the fœtal or the infantile characters of the organ were retained in adult life (*uterus fœtalis, uterus pubescens*). The *complete absence* of the uterus in an individual otherwise female is a very rare occurrence, and the condition diagnosed clinically as absence of the uterus (*uterus deficiens*) is generally one or other of the forms of rudimentary uterus. In all these conditions there are often found to be other defects of the genital apparatus, such as imperforate vagina, rudimentary ovaries, ill-developed mammary glands, and absence of hair on the mons. Clinically there is generally amenorrhœa, but in the fœtal and infantile types there may be a scanty and painful discharge; there is always sterility, and commonly dyspareunia; and should normal ovaries be present there will be so much periodic pain as to necessitate the consideration of the question of their removal. The diagnosis of the anomaly can only be made by a careful vesico-vagino-abdominal examination of the pelvic contents; in the absence of a perforate vagina a recto-vesico-abdominal examination will be required. In the case of the fœtal and infantile uterus attempts to stimulate growth by means of ferruginous tonics, intra-uterine stem pessaries, and electricity, have been made and occasionally have obtained a certain measure of success; but in the more marked forms of the rudimentary uterus the removal of the ovaries is generally indicated if the sufferings of the patient from ovulation are severe.

Congenital anteflexion and *retroflexion* of the uterus have been regarded by some as malformations; it is probably more correct to look upon them as the results of fœtal disease (fœtal peritonitis), or of pelvic peritonitis in childhood. They are attended by menstrual suffering (scanty and painful), by sterility or early abortion, and by various reflex phenomena; and are very rebellious to treatment. *Congenital prolapsus uteri* is a very rarely observed condition; so far all the cases recorded (about a dozen) have been accompanied by lumbo-sacral spina bifida which has been the cause of early death.

The malformations of the vagina in many respects resemble those of the uterus. *Double vagina*, for instance, is due, like double uterus, to want of fusion of the Müllerian ducts. A septum, more or less complete, divides the vagina into two lateral canals (rarely into two canals lying antero-posteriorly). In the least-marked degree there is only a ridge on the vaginal wall or on the cervix to indicate the double nature of the canal. The septum varies much in thickness; it may be absent above and present below (*vagina septa infra*), or present above and absent below (*vagina septa supra*); and it may be perforated in several places. Generally the anomaly is associated with one or other of the forms of double uterus; there may also be a double hymen, but a double vulva is most uncommon, and probably belongs to quite another group of anomalies (double terata). In cases of labour with vagina septa the septum may cause delay and require to be divided, but it may also be pushed aside, and may indeed pass undetected. One of the canals may be imperforate, and mucus may collect in it, giving rise to the erroneous diagnosis of vaginal cyst: when it is associated with a functionating uterus, blood will accumulate in it (*unilateral hæmatocolpos*) and require evacuation.

Unilateral vagina is practically always a concomitant of uterus unicornis. The canal is narrow, and lies somewhat to one side of the middle line; it in fact represents one of the Müllerian ducts, the other having aborted.

Complete absence of the vagina (*defectus vaginæ*) is very rare, and is nearly always associated with advanced teratological conditions, such as sympodia, but incomplete development of the vagina is comparatively common. To this condition the name *vagina rudimentaria* has been given, but a better term is *atresia vaginæ*. There is a complete or an incomplete imperforate condition of the vagina; simply a muscular cord may lie between the rectum and the bladder, or the canal may be present above and absent below, or *vice versa*, or membranous diaphragms may exist in it at various levels. These anomalies are due to imperfect canalisation of the fused Müllerian ducts; but when the lower or vestibular part of the vagina alone is absent it may be supposed that the fault lies, as Hart suggests, in the Wolffian bulbs which normally form the lining of this portion of the canal. The uterus and ovaries may also be imperfectly developed; at the same time this cannot be counted upon, for not infrequently the internal genitals are functionating, and then the condition known as *hæmatometra* (retention of menstrual blood in the uterus) is produced. Clinically, vaginal atresia does not attract notice till puberty, when it is found that menstruation is not established. There may be, however, every month a feeling of weight in the pelvis along with headache, swelling of the mammæ, and epistaxis; and a physical examination may then reveal the absence of the vaginal canal, or (in the less-marked varieties) the presence of a bulging membrane at the vulva. Probably many of the cases of so-called imperforate hymen are really instances of imperforation of the lower part of the vagina, and sometimes the hymen can be seen on the outer aspect of the bulging membrane. By means of rectal touch, aided by a sound in the bladder, the amount of vaginal imperforation can be made out, and the chances of successful surgical interference gauged. It may become necessary to operate, either to allow the escape of retained menstrual blood or to establish an artificial vagina for coition. In the least-marked degree a crucial incision through the membranous obstacle will suffice to set the discharge free; strict surgical cleanliness must, of course, be observed, and means taken to prevent subsequent contraction of the canal. In the more extensive imperforations it becomes very difficult to reach the uterus, and dissection upwards between the bladder and the rectum may be tedious and ultimately futile; the opening of the abdomen in order to operate from the peritoneal aspect has been scarcely more successful. Recently, however, it has been proposed to make straight for the pouch of Douglas, and not to attempt to avoid it, to open into it as in the operation of posterior colpotomy, and then by palpation inside the pelvis to determine whether the uterus and ovaries are healthy. In the presence of functionating internal genitals it will then be well to attempt to form a vagina by invaginating the vulvar mucous membrane, and uniting it with the cervix uteri, and the opening into the pouch of Douglas can then be closed. When the internal organs are not active the gynecologist will be well advised to leave things alone; the demand for an artificial vagina must come from the patient herself, and all the dangers of the operation ought to be pointed out to her and to her husband. By *lateral atresia vaginæ* is meant an imperforate condition of one-half of a septate vagina; blood may accumulate in it and give rise to *lateral hæmatocolpos;* suppuration also may take place in it, *lateral pyocolpos;* in either case the treatment is incision of the sac, with, sometimes, excision of the sac-wall.

Malformations of the vulva do not all admit of so simple an explanation as the anomalies of the uterus and vagina, for they are due to defective development of the posterior end of the embryo, a part whose normal development is not as yet fully understood. The presence of *two vulvar apertures* (one of the rarest of malformations) probably indicates a minor degree of posterior duplicity of the trunk. *Atresia vulvæ superficialis* is not a malformation in the true sense of the term, for it is doubtless due to adhesion of the labia produced by fœtal or infantile vulvitis. It is easily corrected; sometimes simple traction of the labia serves to separate them; in other cases it is necessary to pass a director in at the anterior opening (in the neighbourhood of the meatus urinarius) and cut down upon it. *Vulvar anus* (*atresia ani vaginalis*) is the name given to a condition in which the normal anus is absent, and the rectum opens into the vagina or vulva (or rather into the urogenital sinus, for the anomaly is really a persistence of the early cloacal state). The chief symptom is the passage of fæces, with or without sphincteric control, in the neighbourhood of the posterior commissure of the vulva. Operative interference, which is most likely to be successful if undertaken when the patient is about fifteen years old, consists in passing a probe in through the vulvar anal opening, and bringing it out at a spot corresponding to the normal position of the anus, and then dividing the parts lying between and bringing the rectum down and suturing it in position. Recently it has been suggested to split the fibres of the levator ani in order to gain a good sphincter. *Vulva infantilis* is of importance only as indicating that in all probability the internal genitals likewise are in an undeveloped or infantile state. *Atresia hymenalis* is a malformation which is presumably often met with, for many cases are on record, but there is reason for believing that many of these cases have really been instances of imperforation of the lower end of the vagina. At any rate the symptoms, diagnosis, and treatment of the anomaly are the same as those of the minor degree of vaginal atresia. The *clitoris* may be *absent*, less rarely it is *split;* both these malformations occur in association with epispadias in women or with ectopia vesicæ. *Hypertrophy* of the *clitoris* and *labia* may be met with as a congenital anomaly, and may give rise to local irritation and to more distant reflex phenomena; excision of the enlarged parts will then be indicated. *Adhesion of the clitoris* to its prepuce and to the nymphæ has much the same symptoms as phimosis in the male, and may be cured by the separation of the adhesions. Finally, it may be noted that the *hymen* may be unusually *fleshy* or *tough*, or that it has an abnormal shape (*labiate, denticulate, fimbriated*, or *circular*). When it is tough it may cause dyspareunia or prevent coitus altogether, and when it is abnormally vascular it may lead to the occurrence of serious hæmorrhage from rupture in coition. Sometimes there are two apertures in the hymeneal membrane (*hymen septus, biforis*), rarely there are several (*hymen cribriformis*).

Genu Valgum. *See* DEFORMITIES.

German Measles. *See* RUBELLA.

Glandular Fever.

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GLANDULAR fever is an acute infectious fever characterised by inflammation, apparently primary, of lymphatic glands, beginning in the deep cervical group and sometimes limited to it, and by constipation. The epidemic nature of the disease was first maintained by Pfeiffer in 1889, and German writers frequently refer to it as Pfeiffer's "Drüsenfieber."

The disease attacks children especially, and when introduced into a household few between the ages of six months and thirteen years escape; adults, though not altogether immune, appear to contract the disease much less readily

The incubation period varies from five to ten days, but the most usual period is seven days.

The onset is usually quite sudden, the first complaint being of pain in the neck and headache. The pain is aggravated by movement and the head is held stiffly. It will now be found that the temperature is raised (101° to 103° F.), that the face is flushed, the tongue furred, and that there is deep-seated tenderness in the neck, especially in the anterior triangle, though no enlargement of glands may be perceptible. In many cases abdominal pain is an early symptom, and there may be tenderness on deep pressure. Enlargement of the liver can be made out in most cases, and of the spleen in some. There is no jaundice. The child refuses food, and suffers from nausea or vomiting. There is usually some pain on swallowing, but there is either no acute pharyngitis, and this is the rule, or only some slight catarrh which is insufficient to account for the dysphagia and pain in the neck. Constipation is nearly always present at this stage, but in very mild cases small mucous stools may be passed at frequent intervals. After these symptoms have persisted for two or at most three days, attention is drawn to an oval swelling beneath the sterno-mastoid muscle, and presenting at its anterior border. In almost all cases this swelling is situated on the left side, and is perceived on palpation to be due to two, three, or more glands. Medical aid is often first sought at this stage. The enlargement of the glands continues to increase for two or three days more. At the end of this period the pain in them ceases, and they decrease in size. Meanwhile adenitis has probably begun in other groups of glands, and runs a similar course. The glands most usually affected second in order are the corresponding group on the right side, but the posterior cervical, the axillary, the inguinal, and the mesenteric may become enlarged and tender, and some observers believe that the last-named are involved in at least half the cases. The temperature commonly attains its maximum, it may be as high as 104° F., on the third day, when the adenitis of the first group of glands to be involved is in its most acute stage. Its later course is uncertain, and depends apparently on the number of groups of glands attacked. There is no distinct defervescence so long as fresh groups are being attacked. Convalescence usually begins at the end of a fortnight. The commencement of defervescence is frequently marked in severe cases by the passage of numerous copious green stools containing much mucus.

The pulse continues rapid for a day or two after the temperature falls. The enlargement of the glands diminishes slowly, but eventually it disappears completely. Suppuration seldom or never occurs. The severity of the disease varies very much in different cases. As a rule it is a mild affection, but occasionally the symptoms are severe, and suggest the onset of typhoid fever. Such cases may be complicated by nephritis, and there may be blood in the urine. Convalescence even after mild attacks is generally prolonged. The child is left in a condition of pronounced anæmia, it is languid and disinclined to exertion, appetite is poor, and even under favourable circumstances it may be several months before the health is thoroughly restored.

The diagnosis, as will have been gathered from the above description of the symptoms, must be made mainly by exclusion. The symptoms are such as will call attention to the probability of acute disease of the tonsils or pharynx. The sudden onset, after a day or two of fever, of stiffness of the neck, followed in a couple of days or so by enlargement of lymphatic glands on one side of the neck, is, especially if accompanied by abdominal pain and constipation, very suggestive of glandular fever. Suspicion will be confirmed by the occurrence of other similar cases among children resident in the same house, and it should be remembered that in the same family some cases may be so mild as easily to escape attention, while others may be so severe as to raise a suspicion of typhoid fever.

The pathology of the disease is obscure, and will probably not be cleared up until opportunity has been found for a thorough bacteriological study of an epidemic. It is possible, if not probable, that the primary departure from health is some infection of the intestine; the enlargement of the mesenteric glands observed in a large proportion of cases is held to be secondary to this, and it has been sought to account for the fact that the cervical glands are affected first on the left side by the assumption that the infection is conveyed by the thoracic duct. The resemblance which the disease presents to the mildest form of bubonic plague (pestis minor) and to the "non-venereal bubo" of military writers will not escape attention.

Treatment must be directed to the relief of symptoms, for there appears to be no means of modifying the course of the disease. Thus hot fomentations or belladonna applications will relieve the pain attending the enlargement of the glands which usually commences on the left side of the neck, but will not prevent the subsequent enlargement of those on the right side. Constipation may be relieved by laxatives, or by a dose of calomel, but it soon becomes re-established, and repeated purgation is inadvisable. Some advantage may perhaps be derived from the continuous exhibition of small doses of calomel (gr. $\frac{1}{10}$ to $\frac{1}{3}$ or $\frac{1}{2}$) three or four times in the twenty-four hours, and antipyrin is of use to relieve the headache. Sodium salicylate is probably the most useful drug in the early stage when the temperature is high and the pain and stiffness in the neck severe. While the temperature is raised, the child should be kept in bed and receive only fluid nourishment. After convalescence has commenced, the patient should have as much fresh air as possible, but should be warmly clad, especially about the abdomen. Iron and tonics are indicated, and change of air is often necessary to obtain complete restoration to health.

LITERATURE.—1. PFEIFFER, E. *Jahrbuch f. Kinderheilkde.* Bd. xxix.—2. VON STARCK. *Ibid.* Bd. xxxi.—3. WEST, J. PARK. *Arch. of Ped.* vol. xiii. p. 889. Further reference will be found in paper by the writer of this article in the *Lancet*, 1897, vol. i. p. 160.

Glanders (Farcy).

SYNONYMS.—Gk. μᾶλις or μῆλις; Lat. *Malleus, Equinia;* Ger. *Rotz* (*Wurm*); Fr. *Morve* (*Farcin*); It. *Morva* (*Farcino*); Span. *Muermo.*

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I. DEFINITION. — Glanders is a specific infectious disease caused by the *Bacillus mallei*, usually affecting the horse, mule, or donkey, and at times communicated to man and other animals. Where the disease is localised in the nose or internal organs it is called glanders, the cutaneous form being called farcy. The disease runs an acute, subacute, or chronic course.

II. HISTORY.—Glanders has been known from ancient times as a disease affecting horses. It was known to Aristoteles and Hippocrates the Hippiatrist, as also to the Roman writers Apsyrtus and Vegetius (1370-90), the last describing "Malleus humidus" and "farciminosus." Its contagiousness was recognised in the seventeenth century. Solleysel (1664) considered that it could be conveyed through the air, van Helmont (1682) considered syphilis identical with glanders. Saunier (1734) gives directions for disinfecting stables, whilst Garsault (1741) and Bourgelat (1764) recommended the immediate destruction of glandered horses together with the isolation of those suspected. Abildgaard and Viborg in Denmark at the end of the eighteenth century proved that glanders could be communicated by secretions or pus of diseased animals, less frequently was the virus contained in their blood. Virus which had been dried or exposed to a temperature of 45° C. was innocuous. Similar results were obtained in England by Coleman and Delabère-Blaine. Rayer (1837) proved that glanders was communicable to man. The first bacteriological investigations were made by Zürn and Hallier, Christot and Kiener (1868), subsequently by Bouchard, Capitan, and Charrin (1881-82), but it was not until 1882 that Löffler and Schütz isolated the bacillus, and proved finally that glanders or farcy is a specific infectious disease caused only by the *Bacillus mallei*. The last three French investigators had succeeded in cultivating the bacillus from a man and a horse, and, after several generations in culture, successfully infected donkeys, cats, and guinea-pigs. Working with fluid culture-media, they concluded that the specific agent was a coccus growing in chains. O. Israel (1883) obtained cultures on blood-serum from horses, and successfully infected rabbits with the bacillus, whilst Kitt (1883-84) and Weichselbaum (1885) confirmed and extended the knowledge gained.

III. GEOGRAPHICAL DISTRIBUTION AND PREVALENCE.—Though glanders is not as prevalent as it used to be, nevertheless it is the most dangerous equine disease. It would appear as if glanders can occur in any climate, but it certainly exhibits a predilection for certain localities. Its gradual increase as we go south (the reason of this is unknown) is evident from the figures given by Krabbe for 1857-1873 per 100,000 horses; Norway 6, Denmark 8·5, England 14, Wurtemberg 77, Prussia 78, Servia 95, Belgium 138, France 1130, Algiers 1548. During the years 1877-1887 there were 4 per 100,000 in Sweden (Lindqvist), 300 per 100,000 in Austria. During 1884-1897 in Germany the frequency was 20-30 per 100,000. In European Russia glanders is very prevalent, being estimated at 400 per 100,000. In 1898 there were 1380 horses attacked in England (almost entirely in metropolitan districts), 138 in Belgium; there were 657 outbreaks in France and 17 in Denmark. In Hungary the loss from glanders was 39 per 100,000 in 1897; and in the same year Italy lost 277, and Switzerland 58 horses (dead and killed). Sanders (1896) says glanders has been known but recently in South Africa. Dávalos (1894) says it was introduced into Cuba in 1872 (9 cases between 1888 and 1893). Penna (1898) reports it from the Argentine Republic, and v. Velzen from the Dutch East Indies. It also occurs in the U.S. and Canada.

IV. Animals affected.—The disease occurs spontaneously in the horse, mule, donkey, and at times in menageries (lion, leopard, tiger, bear). A number of other animals have, however, been found susceptible to experimental infection. The difference in the statements of authors regarding the degrees of susceptibility of certain animals to inoculation would seem to depend in part upon variation in the virulence and the amount of the culture used. Highly susceptible are field-mice, *Arvicola arvalis*, *A. terrestris* in Europe, *A. riparius*, *A. austerus* in America, *Spermophilus guttatus* and *S. Citillus* in Eastern Europe, *Spermophilus Townsendi* and *S. Richardsoni* in America, the cat, guinea-pig, hedgehog (*Erinaceus europeus*). White and house-mice are more resistant, though susceptible to virulent cultures. Rabbits, the goat, sheep, camel, and young dog have been successfully infected. The resistance of adult dogs has been overcome by inoculating cultures whose virulence had been raised by intracerebral inoculation in another dog. Young pigs have been successfully infected by inoculation into the anterior chamber of the eye or into the lungs, but not through the skin. Adult pigs, unless weakened, are absolutely resistant, as are also cattle (including the calf), chicken, pigeon, linnet, and frog (Löffler, 1886; Kitt, Weichselbaum, 1885; Csokor, 1888; Peuchu, 1889; Straus, 1889; Preusse, Grünwald, Salmon, Sanarelli, Kranzfeld, Cadeac and Malet, Tartakowski, Dschunkowski, 1886-99).

V. Bacteriology.—The *Bacillus mallei* is found in the affected tissues and organs and their secretions, as also the discharges from ulcers, etc. They may be very generally distributed in the body. In man, the horse, sheep, guinea-pig, and rabbit, they have been isolated and cultivated from nodules, cutaneous abscesses, the greatly enlarged skin follicles, ulcers of the mucous membranes, more rarely from marrow and blood. They have been found in the saliva and milk, but still oftener in the urine secreted by kidneys showing no appreciable lesion. In man they have been found in the abscesses and blood (few) during life. In the horse they may appear in the blood during a febrile attack or after a mallein injection. They are more numerous in the blood in acute cases. It is often difficult to find the bacilli microscopically in the nodules, but their presence may be demonstrated by cultures or inoculations upon animals (Löffler, Weichselbaum, Ferraresi and Guarnieri, Lissitzin, Preusse, Babes, Hallopeau and Jeanselme, Bonome, Duval, Gasne and Guillemot, Noniewitz, Kitt, Trambusti, Philipowitcz (1886-98).

Morphology.—The form of the glanders bacillus varies greatly. The bacilli measure 1·5-3 by 0·25-0·4 μ. They have rounded extremities, and are often slightly curved. Noniewitz (1891) claims to have observed differences in the form of bacilli within the body. In acute cases well-formed bacilli are encountered, in subacute and chronic cases more highly refractive coccus-like bodies are found, which suggests that many of them degenerate in the body. Gutzeit (1892), as also Johne, finds the bacilli are larger when grown in horse-bouillon than when cultivated in beef-broth, Already Löffler observed club-shaped, filamentous, and coccus-like forms in culture. and this has since been seen and described by Kranzfeld, Levy, Semmer, Marx, and Galli-Valerio (1887-99); the two latter, as also Conradi (1900), have in addition observed true branching in organisms grown in culture. Lubarsch (1899) was unable to observe branched actinomyces-forms in rabbits which had been inoculated into the kidney or subdurally. The glanders bacilli do not form spores. Refractive bodies which have been considered to be spores by Baumgarten (1888) are almost certainly degenerative. These bodies show no greater resistance to heat and chemical agents than do the ordinary bacilli, and nobody has claimed to see bacilli issue from them. The long thread-like forms may measure 10-26 μ or more. The bacillus is non-motile.

Staining Reactions.—Smear preparations should be made as thin as possible. The bacillus stains readily with basic aniline dyes, presenting usually irregularities, *i.e.* alternate deeply staining or clear and faintly staining parts. Bacilli are best looked for in young nodules. Owing to the fact that they readily decolorise and are often hidden by the nuclear chromatin contained in the necrotic tissue in which they lie (Unna) it is at times very difficult to demonstrate them in sections. They may be demonstrated in smear-preparations by means of Löffler's alkaline methylene-blue (methylene-blue conc. alcoholic sol. 30 ccm., aqueous solution of caustic potash ($\frac{1}{10000}$) 100 ccm.), the stain acting for 5 minutes, after which the preparation is dipped for 1 second in 1 per cent aqueous solution of acetic acid to which a few drops of an aqueous solution of tropäolin 00 have been added, after which they are quickly washed off with distilled water, dried, and mounted in balsam. Tropäolin decolorises the protoplasm of the cells and their nuclei (partially), but not the bacilli (Löffler). Kühne (1888) advises to transfer sections from alcohol to water, staining with carbolic methylene-blue for 3-4 minutes, decolorising a few seconds with acidulated (HCl) water, washing thoroughly with water, and drying by pressing down upon the section with blotting-paper. The section is then treated 8-10 minutes with aniline oil, to which 20 per cent oil of turpentine has been added, this being followed by oil of turpentine, xylol, and mounting in balsam. Sections thus prepared do not show histological details, and are not durable. Unna (1893) directs to dry the sections upon the slide, stain ½ hour with methylene-blue (according to Löffler, Kühne, Unna); place the slide in water, thus liberating the section, which is then put for some seconds in glycerine-ether mixture, and thence backwards and forwards to water, finally through water, absolute alcohol, oil of turpentine, and mounted in balsam. For double staining he recommends placing the sections in 1 per cent acid fuchsin overnight, washing with water, drying, staining ¼ hour in methylene-blue, followed by treatment with 1 per cent arsenious acid for 5-10 seconds, water, alcohol, oil of Bergamot, and mounting in balsam. Abbott (1899) finds the following simpler method useful:—Transfer the sections from alcohol to distilled water, thence to dilute fuchsin kept 15-20 minutes at about 50° C. Transfer to a slide, absorb superfluous stain with blotting-paper, treat ½-¾ minute with 1 per cent acetic acid, remove the acid with distilled water, dry the sections with blotting-paper, wash it 15 seconds with absolute alcohol, clear with xylol, and mount in xylol balsam. Muir and Ritchie (1899) especially recommend carbol-thionin-blue. Add one part of stock-solution, 1 gramme thionin-blue dissolved in 100 ccm. carbolic acid solution (1 : 40) to three parts water, and filter. Stain for 5 minutes or longer, wash thoroughly with water, decolorise with weak acetic acid, wash with water, dehydrate with aniline-oil. The bacilli do not stain by Gram's method. Rosenthal (1888) was able to stain bodies resembling spores in bacilli obtained from old cultures when using Neisser's method for staining endogenous spores. But this is no proof that spores actually occur. Babes (1891) finds that the so-called chromatic portions of the bacilli take on a violet colour with Löffler's stain. Stained with aniline-fuchsin, followed by methylene-blue, the bacilli often show bluish red oval granules within their substance. Marx (1889) finds that the clear spaces observed when staining with carbol-fuchsin may be stained brown by Neisser's stain for *B. diphtheriæ* (methylene-blue and vesuvin), this stain also demonstrating 3-5-9 round dark blue bodies (Babes-Ernst bodies) situated at regular intervals within the bacillus.

Cultivation.—Though pure cultures have been obtained from abscesses they are best made from fresh nodules, as these contain most bacilli. It is best to make the first culture upon potato or blood-serum, as negative results are at times obtained if they are immediately transferred to agar. There are differences observable in the initial cultures which might lead one to suppose that there are different varieties of the bacillus. On potato some rapidly assume their characteristic colour, others do so slowly, whilst some grow well and others badly upon gelatin. Passage through the guinea-pig renders the germ easier to cultivate (v. Babes, 1891). Bacteriologists all agree that the bacillus shows its most characteristic growth upon potato, and, as it grows excellently upon this medium, it is well for purposes of diagnosis to cultivate initial cultures thereon. The bacillus grows best in the presence of oxygen, and at a temperature of 35°-37° C. It grows fairly well at 25°, very slowly at 21°, which may be considered the minimum temperature at which it will multiply. It still grows at 42°, but not at 43° C. It grows better upon *slightly acid or neutral media*, and preserves its virulence longest upon acid potato.

Potatoes are best prepared, according to Kresling (1892), by washing the slices with 0·5-0·7 per cent sodium carbonate solution until the wash-water remains clear. The potatoes are sterilised 80 minutes at 110° C. They should have a degree of acidity corresponding to 0·1-0·3 ccm. of $\frac{1}{10}$ normal soda solution. Cultures should be prevented from drying. The bacilli will not grow upon potato at room-temperature, being best maintained at 36°-36·5° C. (Marx, 1899). Initial cultures show a brownish, moderately thick, and slimy growth after 3-5 days, at times earlier. Subcultures grow more rapidly, the growth presenting a clear, honey-like appearance, subsequently growing dark and opaque, acquiring a *café au lait* or chocolate colour in the course of a week or so. The potato about the edge of the growth often exhibits a greenish discolorisation. Mashed potatoes spread in a layer upon the bottom of Erlenmeyer flasks also afford an excellent means of cultivating the germ. Next to potato, blood-serum and then glycerinated agar are the best media. On coagulated or gelatinised *serum* after 3 days at 37° small transparent, discrete drop-like colonies appear. Upon *glycerin-agar* slants it forms a somewhat slimy, grayish white translucent growth, which with time acquires a brownish tint. Whereas it grows badly on white of *egg* it grows well upon sterilised yolk, forming knob-like growths after 24 hours (Marx, 1889). It also grows well upon sterilised *carrot*, forming a white pigment in 2-3 days. They form a stringy white mass in gelatin, which takes some weeks to become liquefied. They grow well in glycerinated *bouillon* which has not been neutralised, at first uniformly clouding the medium, and subsequently forming a stringy deposit. Babes (1891) recommends agar made with potato juice instead of meat infusion as a culture medium. Milk is coagulated in 10-12 days at 37° C., the reaction being neutral (Gorini, 1896). It grows in deep gelatin at 22°, and in a hydrogen atmosphere at 37°, but not as well as under aerobic conditions (Marx, 1899).

Products.—We have already referred to the pigment which is formed by the bacillus, and stated that it constitutes an important characteristic of the organism. The pigment varies somewhat, depending upon the nature of the medium. On acid potato it is brown, on agar a pale straw colour, in bouillon at times orange-coloured, on carrots white (Smith, 1889; Marx, 1899). The bacilli exert a pathogenic effect by virtue of toxic substances which they give off or contain. If dead bacilli are inoculated into an animal they produce local pathogenic effects. If the bacilli have first been washed with distilled water they exert less effect. If introduced in sufficient quantity they produce a cachectic condition which ends in death (Babes, Rigler, and Podasca, 1897). Kalning, Preusse, Pearson, Roux, and Foth (1891-96) have by different procedures obtained "mallein" from cultures of the glanders bacillus. Mallein is a compound similar to "tuberculin," and constitutes from a practical point of view the most important product of the germ. Kalning obtained it from potato cultures which he rubbed up with water, heated to 120° C. for 20 minutes on four successive days. He kept the fluid for two days at 39°, and then passed it through a Pasteur filter. Preusse used old dried-up potato-cultures, rubbing them up with glycerine and water, and placed them some days in the thermostat, after which they were filtered and sterilised, the filtrate being the mallein. Roux concentrated the sterilised filtrate of glycerin-bouillon cultures a month old, obtaining a syrupy fluid, which had to be diluted with ten times its volume of ½ per cent carbolic acid for use. The dose of concentrated mallein for a horse amounts to ¼ ccm. (Nocard, 1895). The value of mallein in diagnosis has been tested thousands of times upon horses, and in a few cases in man. It is without doubt invaluable in diagnosis, especially of hidden glanders in the horse. Properly prepared it preserves its qualities unimpaired for 1-2 years. Foth (1893-94) has made a dry preparation for which he claims greater stability. (See further under Diagnosis.) De Schweinitz and Kilborne (1894) consider that the active principle of mallein is an albumose which can be precipitated from cultures by alcohol or ammonium sulphate. Its chemical composition has been investigated also by Kresling (1892) and Gutzeit (1892), but our knowledge is still unsatisfactory. Levandowsky (1890) found cultures of the bacillus in bouillon to contain indol and phenol, these according to Kresling (1892) being absent in potato cultures. The bacilli also contain a peptonising ferment, as is seen from the liquefaction of gelatin.

Chemical Composition.—When the bacilli are dried at 110° C. they yield dry substance equal to about 24 per cent of their original weight. The ash (6·67 per cent) contains much phosphoric acid, potassium, sodium, sulphuric acid, and traces of iron and chlorine. The nitrogen amounts to 10·5 per cent. The dry substance is soluble as follows: in water 25·75 per cent, in ether 2·84 per cent, ether and alcohol 3·2 per cent, alcohol 0·66 per cent, the rest remaining insoluble in any of these agents. The ethereal extract yielded a yellow fat melting at 40° C., and containing lecithin, cholestearin, and oleic acid (Kresling, 1892).

Behaviour of the Bacilli outside the Body.—Löffler (1886) did not believe that the bacilli would multiply outside the body under ordinary conditions. They do not do so in hay and other infusions. Bacilli obtained from different sources vary considerably in their powers of resistance. Cadeac and Malet (1886) found that they could not withstand drying; Bonome (1894) found they died after 10 days at 25°, whilst dried over sulphuric acid they only died after 35 days. Under the most favourable conditions Löffler found that they did not retain their virulence longer than four months. Sanarelli (1889) found them virulent longest (two months) in anaerobic cultures, in aerobic cultures they lost their virulence in a few days. They have been found to resist putrefaction for 14-30 days, to survive 15-20 days in water. When kept in a moist state at 23° they were still alive and virulent after 20 days, whereas they had died within five days at 15°-16°. They are killed by a $\frac{1}{2000}$ sublimate solution in one hour, by turpentine water ($\frac{1}{100}$) in one hour, by carbolic acid (5 per cent) in five minutes, etc.; in other words, they are readily destroyed by the ordinary antiseptics. When exposed on threads to sunlight they are destroyed in one day. The statements of authors with regard to the effects of heat vary considerably, this being evidently due to the different conditions under which the experiments were carried out. When heated for one minute at 62°, or ten minutes at 55° in culture media, they are killed (Marx, 1899). Bonome (1894), however, found them alive when heated to 70° degrees for six hours in a moist state, though they were killed at 70°-75° in five to six minutes, at 90°-100° in three minutes. Finally Bromberg (1891) found that all the bacilli in old cultures were not destroyed by half an hour's exposure at 100°. (See also Sirena and Alessi, 1892; Plemper van Balen, 1897.)

Mode of Infection.—Glanders infection may usually be traced to direct contact with diseased animals, whose discharges from the nose, as also pus, urine, saliva, and milk, have been found to contain the bacilli. The secretions of ulcers and nasal discharge are particularly dangerous. Infection may take place indirectly through harness, blankets, etc. that have been in contact with glandered animals and become soiled with diseased secretions. In many cases a lesion of the skin serves as a port of entry. Babes (1888) considers that infection may occur through the uninjured skin, having succeeded in infecting guinea-pigs by applying a mixture of bacilli in vaseline to their skins. The bacilli penetrated into the hair-follicles, and passed thence through the epithelial layer into the lymphatics. Cornil and Nocard (1890) obtained a similar result in two out of fifteen experiments. V. Babes (1893) cites two cases in man where infection seems to have occurred through the intact skin. It has been claimed, but remains unproved, that infection may occur through blood-sucking flies. Infection has been produced experimentally through inhalation of dried bacilli, and the nasal mucous membranes and lungs may be primarily affected. Lesions in these situations are, however, frequently due to secondary infection, the bacilli having reached the part from primarily affected organs elsewhere. The nasal mucous membrane offers an especially favourable nidus for the multiplication of bacilli carried thither by the blood-currrent. Friedberger and Fröhner (1896) consider that in nine-tenths of the cases in horses infection occurs through inhalation, but this view is rendered untenable through Nocard's (1894-97) exact experiments proving the readiness with which they may be infected through feeding. Nocard considers the intestinal tract to be the chief port of entry. If pure cultures of the glanders bacilli are mixed with the food or drink of horses, donkeys, rabbits, or guinea-pigs, infection is certain to occur. At autopsy numerous minute translucent or even calcified nodules are observable in the lungs. The negative results obtained in such experiments by Cadeac and Malet (1894) may be explained through their using less virulent cultures than did Nocard. Infection through feeding has certainly occurred spontaneously in dogs, cats, and animals in menageries. Infection of the fœtus occurs in a limited number of cases in the horse, dog, and guinea-pig (Löffler 1886, Cadeac and Malet 1886, observed this twice in thirteen experiments; Bonome 1894). A few cases of infection have occurred in bacteriological laboratories, once through the prick of a hypodermic needle whilst inoculating an animal. Persons who have to do with horses are naturally most affected, nevertheless some cases have been observed where no such connection could be traced. Zawadzki, and Nencki and Pruszynki (1896) report the case of a physician who infected himself whilst operating upon a fellow-practitioner who also subsequently died of glanders. Veterinarians have become infected through being bitten whilst examining a horse's mouth, or through secretion being thrown upon the apparently intact conjunctiva. Rémy (1897) has collected nine cases of man to man infection. He cites the case of a woman who became infected whilst making horse-hair mattresses, as also that of a person who acquired glanders from being struck by the fist of a man who owned a glandered horse. Grawleswki (1893) cites the case of a washerwoman who developed a glanders pustule three days after washing the clothes of a man who had died of the disease. He also tells of an old man who attended a person who died of glanders, and developed initial lesions in places where he was accustomed to scratch himself. Tessier (1852) and Cooper (1887) made observations which indicated that infection may occur in man through coitus. Gibert (1840) and others since have proved the auto-inoculability of glanders in man and animals. It is evident, then, that a man may infect himself in other parts of his body through scratching himself.

Increase and Decrease of Virulence.—The bacillus rapidly loses its virulence in cultures, so that it is necessary from time to time to pass it through a susceptible animal. Gamaleia (1890) was able to increase the virulence by continued passage through Spermophilus, and Foth (1896) did this with field-mice. Tedeschi (1893) did this through intracerebral inoculation of susceptible animals, being able to kill dogs with the more virulent bacilli. Sacharow (1893) raised the virulence for cats by repeated passage (eight) through this species, but found then that the bacilli had lost their virulence for horses, a fact which has been observed in other germs. Straus (1889) claimed that they became attenuated by passage through the wolf. It is certain that the bacilli vary greatly in virulence even when taken directly from the horse (Kitt, 1896), and tested upon horses and guinea-pigs.

Experience in Russia, as also observations reported by Semmer (1894) in Germany, prove the existence of a mild form of glanders. This milder form is often referred to as "southern glanders," because of its greater frequency in France, Italy, and Southern Russia. It seems to be unknown in Northern Russia, Germany, and Scandinavia. Whether this depends upon a virus of lessened virulence or upon an inherited resistance of certain southern and eastern races of horses is not determined.

VI. IMMUNITY.—We have noted above the immunity or susceptibility shown by certain species of animals towards this disease. There exists also an individual immunity or susceptibility. It has, for instance, been repeatedly observed that certain horses could be exposed with impunity for months or even years by being kept in stables with glandered animals. This natural resistance may, however, be overcome through overwork, bad food, neglect, poor stabling, exposure to cold or the debilitating effects of intercurrent disease. This explains the greater prevalence of glanders amongst horses in times of war. One attack of glanders seems to convey a certain degree of immunity, but the evidence can scarcely be termed satisfactory. Experiments at producing immunity through inoculation with dead or attenuated cultures, as also with the serum of naturally immune animals, or such as have recovered from an attack, or been rendered resistant to increasing doses of the virus, as also inoculations with mallein, have not given fully satisfactory results. Some investigators report positive results, others negative ones, others again that they have obtained inconstant results. (Straus 1888, Charrin 1885, Finger 1889, Sadowsky 1891, Chenot and Pick, Semmer 1892, Sacharow 1893, Semmer 1894, Hüppe, Schindelka, Bonome, Schattenfroh 1894, Preusse, Nocard, Comeny, v. Babes, Rigler, and Podaska 1897.)

Since masked glanders has been more often detected through the use of mallein in diagnosis, it has been made quite clear that glanders is by no means the fatal disease it used to be thought. Faverot and Humbert (1893) state that as long as the nodules are gray and transparent it is possible for the animal to overcome the bacilli; when the nodules undergo cheesy degeneration the bacilli usually overcome the animal. Animals kept under good hygienic conditions stand a much better chance of recovery. As this was determined upon animals but slightly affected, mallein being used for diagnosis, it does not alter the prevailing opinion based upon well-marked clinical appearances, for the more developed disease is practically incurable. Nocard (1896, 1897) holds strongly to the opinion that recovery may result in quite fresh cases, for the reason that he has frequently observed spontaneous recovery under ordinary conditions as also in experimentally infected animals. Rémy (1897), who has gone over the literature, cites twenty-three recorded cases of recovery from glanders in man between the years 1812-92. Whilst acute glanders or farcy may lead to a fatal termination in 8-15 days, the chronic form may last for months or even years. In chronic cases death may be caused by pyæmia or acute glanders. Hallipeau and Jeanselme report the case of a man who suffered six years. The disease was characterised during the first three years by recurring phlegmons and abscesses, but no general symptoms, the lesions healing spontaneously, or as the result of treatment with caustics. A latent period of three years now followed, when the disease broke out again in the acute form, the man dying. Bollinger observed a case of eleven years' standing.

M'Fadyean (1896), and subsequently Foulerton, Wladimiroff, Bourges and Méry (1897-98), observed that the serum of a glandered animal agglutinated bouillon cultures of the glanders bacillus. Semmer (1892) was unable to produce immunity in cats and guinea-pigs through the serum of naturally immune animals such as the horse and cattle, whilst Chenot and Picq (1892) claimed they had been able to save seven-tenths of infected guinea-pigs by the use of cattle serum. When guinea-pigs had been inoculated with very virulent cultures (which killed in five days) the serum retarded death until the 21-42 day. V. Babes, Rigler, and Podasca (1897) state that donkeys treated with increasing doses of mallein, and finally with injections of dead bacilli, yield a preventive and curative serum. Semmer treated horses with mallein and found their serum only conferred a transitory immunity.

VII. SYMPTOMS.—*In the horse* acute glanders has a period of incubation varying from 3-8 days or longer. At the onset febrile symptoms manifest themselves, the horse loses its appetite, and is sluggish. In a day or two a serous nasal discharge appears, which at first may be from one nostril only; by the fifth or sixth day this discharge is purulent, yellow, and viscid. The margins of the nostrils are swollen and the lymphatics of the face inflamed. The nasal mucous membrane, at first hyperæmic, inflamed, and studded with small nodules, later shows large irregular ulcerating patches, which tend to become confluent and form large serpiginous ulcers with raised edges. These ulcers may eat into the deeper tissues, and lay bare and erode the cartilage of the septum and the nasal bones. The submaxillary lymphatic glands are enlarged, indurated, and in some cases become adherent to the surrounding tissue; they are not tender and seldom ulcerate. Hot, painful œdematous swellings appear on the trunk and limbs; if on the latter they cause lameness. The disease reaches its height in from 6 to 15 days, the animal being by this time in great distress. Breathing is snuffling and difficult, the animal constantly snorting to expel the mucus and viscid nasal discharge which glues together the alæ nasi. The lymphatics of the face and other parts of the body are often inflamed and corded (acute farcy), and death from suffocation preceded by painful dyspnœa follows rapidly.

Acute Cutaneous Glanders: Farcy.—The onset resembles that of acute glanders, and as a rule acute glanders is present at the same time; if not, it usually supervenes before death. The characteristic local lesions are inflammation of the lymphatic vessels, which appear as firm cords running under the skin, and the formation of nodules situated at intervals along these cords. The nodules show a marked tendency to ulcerate and discharge a thick oily material. These changes may appear in any part of the cutaneous surface, but show a preference for the hind limbs. The ulcers form about the sixth day. They have irregular raised borders, with a yellowish base covered with a grumous discharge. Death follows rapidly, usually from acute glanders, involving the respiratory tract.

Chronic Glanders.—This form of the disease often comes on insidiously; there is frequently some constitutional disturbance, which may subside as the local lesions are developed. The premonitory symptoms vary much. The general health may appear good, but there is some dulness of the eye and roughness of the coat, the animal shows lack of endurance and sweats easily. Lameness, intermittent or continuous, may be noticed, there may be œdema of one or more limbs, cough, bleeding from the nose, and possibly orchitis. The nasal discharge often present is at first clear, but eventually becomes cloudy, purulent, and viscid, and the animal is continually snorting to expel the secretion. If the nasal mucous membrane is examined, nodules and ulcers can usually be seen, and after the disease has existed some time stellate scars are also present. The submaxillary lymphatic glands may be enlarged, but as a rule do not ulcerate. The larynx and trachea may be the seat of the primary lesion, the nasal symptoms being delayed. In these cases, cough with varying expectoration, enlarged glands, tender larynx, fœtid breath, and general ill-condition are usually present. The skin (chronic farcy) may be attacked before the respiratory passages. Nodules form and ulcerate, discharging a thick oily material, and generally after a few weeks heal, leaving a dense stellate cicatrix, fresh nodules forming in adjacent or more distant parts, these again healing, to be followed by the formation of fresh nodules. The lymphatics leading from the affected parts are corded (farcy pipes). This process may go on for months or years, the animal either recovering or dying from an acute attack.

Symptoms in Man.—The period of incubation is from 3 to 8 days, but apparently it may extend to three weeks. The disease commences with malaise, headache, vague pains in the muscles and joints simulating rheumatism. If the bacillus has entered through a wound, this may completely heal, but in a day or two the seat of inoculation becomes painful and swollen, an eruption of vesicles often appears around it, and the lymphatics become swollen and painful. Frequently the skin about the seat of inoculation resembles phlegmonous erysipelas. In other cases gastro-intestinal symptoms are most prominent, and the disease simulates typhoid fever. Occasionally a pustular eruption, not unlike that of small-pox, occurs on the face and limbs. In most cases the general symptoms rapidly increase in severity, the joint pains becoming more severe, and œdematous swellings appear on the limbs. The temperature is raised, and though at first the rise may be intermittent, it soon becomes continuous. It is accompanied by severe headache, great prostration, dry skin, foul mouth, and fœtid breath. The pulse is full and soft, varying between 90 and 100. Epistaxis often occurs. Hard, painful nodules may appear in the skin; these soon soften, break down, and form extensive, deep, irregular ulcers which may even lay bare the bone. The affections of the mucous membrane of the nose appear to be less frequent in man than in the horse, but they do occur. There is at first a feeling of dryness of the mucous membrane, accompanied by pain and tension at the root of the nose. The skin over the bridge of the nose becomes swollen and reddened, the mucous membrane is much injected and swollen, a fœtid secretion appears, at first clear and thin, but soon becoming yellow, purulent, and viscid, and finally blood-stained. The eyelids and conjunctivæ are swollen and œdematous, often completely closing the eye. The conjunctiva secretes a purulent fluid. The inflammatory process is apt to spread, invading the pharynx and palate.

The gums are swollen and bleed readily, the breath is fœtid, swallowing is difficult, and the voice hoarse. The lungs are also involved, severe pleuritic pains are complained of, and there is a troublesome cough. Towards the end œdema of the glottis may supervene. The bowels, at first obstinate, become relaxed, and profuse diarrhœa follows. If the liver is involved it is enlarged, painful on pressure, and there may be jaundice. As the disease progresses sleeplessness and nocturnal delirium ensue. If nodules form in the muscles, rigors simulating those of pyæmia are a prominent symptom. Towards the end the heart fails, its beats become quickened, the pulse becomes thready, easily compressible, the temperature rises to 105°-106° F., the skin is covered with a cold clammy sweat, consciousness becomes cloudy, the evacuations are passed involuntarily, and eventually the patient becomes comatose. The extremities are cold, gangrene and ulcers extend, giving rise to a fearful stench, and finally, after complete loss of consciousness and sensibility, dyspnœa increases, the breathing becomes stertorous, and tetanic convulsions close the scene.

Chronic Glanders.—This form of the disease may last months or even years; the patient complains of a vague feeling of discomfort, pains in the extremities and joints, loss of appetite, and sleeplessness. The attacks of fever are recurrent. The symptoms vary in severity from time to time, and eventually either pass into an acute form, or complete recovery may take place. The local lesions in these cases vary greatly. If the skin is the seat of the lesion the surrounding parts become swollen and œdematous, the nodules usually appear in one part only; they break down, leaving indolent ulcers, which may heal, leaving a scar. Abscesses form around joints, and fistulous openings, which show little tendency to heal, appear upon the surface. They discharge a thin foul pus. An eruption of papules, which become pustular, may appear. Affection of the nasal septum is often absent, when it appears it follows a similar course to that met with in the acute disease, but is much more chronic. Extensive ulceration occurs, leading to destruction of the septum and even gangrene of the root of the nose. Inflammation and ulceration of the pharynx and soft palate may occur, and in these cases the lesions strongly resemble those found in syphilis.

VIII. Pathology.—The lesions which occur in this disease are due to the *B. mallei* and its products. This organism lodging in the tissues gives rise to a series of changes, the most characteristic being the formation of proliferative nodules in various situations, chiefly in the mucous membrane of the respiratory tract, lungs, and skin. But they are also found in the subcutaneous and submucous tissue, liver, kidney, dura mater, periosteum, and synovial membranes. These nodules show a marked tendency to break down, forming ulcers if situated on free surfaces.

In the horse a common situation for these lesions is the nasal mucous membrane. Within two or three days of infection a small injected area appears, in which a yellow nodule is seen projecting from the free surface. This yellow centre breaks down and leaves a small ulcer. The nodules usually occur in groups, and the ulcers running into each other may soon form a large ulcer, with irregular indurated edges, a deep infiltrating base. This is surrounded by many small nodules in various stages of development and softening, which, breaking down, increase the area of the ulcerated surface. The ulcers at first discharge a thin glairy fluid; this rapidly becomes purulent and viscid. In addition to these changes the mucous membrane is often swollen and injected, and secretes a viscid mucus which appears to possess specially irritating qualities, leading to superficial ulcerations of the mucous membranes in situations where no nodules are found.

The ulceration, very extensive and destructive, may lead to perforation of the septum and necrosis of the nasal bones. Characteristic nodules, at first translucent, hard and shotty to the touch, form throughout the lungs. These are surrounded by an area of consolidated lung which microscopically shows a proliferation and infiltration of the walls of the air vesicles and exudation of leucocytes, with the formation of fibrin in the air vesicles themselves. The area is usually surrounded by a zone of injection, which is especially well seen on the pleural surface. In chronic cases the nodules become fibrous and even calcified. In acute cases the submaxillary lymphatic glands are enlarged and indurated, and tend to become adherent to the surrounding structures. In the skin the lesions usually run a more chronic course; nodules, diffuse swellings, and inflammation and enlargement of the lymphatics leading from the part occur. The nodules break down and form ulcers and sinuses, which show but slight tendency to heal. In the more chronic cases the ulcers both on the nasal septum and in the skin may heal, leaving dense stellate scars. Hot, painful, œdematous swellings are frequently seen.

In man the process is usually very acute. If the septum nasi is involved, the formation of nodules occurs as in the horse, but the ulceration in this case is usually more rapid, and the destruction of the deeper tissues more extensive; perforation of the septum and necrosis of the nasal bones often occurring within a few days of onset. Ulcers also form on the soft palate and pharynx. The antrum and sphenoidal sinuses may be also involved, and occasionally the inflammation spreads into the cranium, causing inflammation of the meninges. If the lungs are attacked numerous nodules are found scattered throughout their substance. These are small, and surrounded by an area of consolidated lung tissue with a bright zone of injection. If cutaneous lesions are present they commonly occur as papules, which rapidly become pustules, containing a cheesy material or thick, blood-stained pus. A red scab forms, which, if removed, leaves a small ulcer. Œdematous swellings also occur, and in many cases show a striking resemblance to phlegmonous erysipelas. The muscles may contain nodules which caseate; these also frequently occur in the periosteum, especially at the insertion of tendons. In acute cases the spleen is usually enlarged and soft. The liver shows cloudy swelling, and may contain nodules scattered throughout its substance. Similar changes are described in the kidney. Secondary infection may occur, more especially in chronic cases.

IX. DIAGNOSIS.—The diagnosis will depend upon the clinical appearances already referred to and the finding of the specific bacillus, the positive reaction with mallein and the Straus test.

Straus Test.—Dilute the pus, secretion, or juice of diseased gland in sterile water or bouillon, and inject the fluid respectively into the peritoneal cavity and subcutaneously into male guinea-pigs. If impure material is injected (nasal discharge) it is inadvisable to trust to one guinea-pig, as it may die of suppurative peritonitis before the characteristic orchitis has had time to develop. Where the guinea-pig is inoculated with pus containing few bacilli the orchitis may be late in appearing. Levy and Steinmetz (1895) advise to make the intraperitoneal injection in the median line above the bladder, so as to avoid injuring the naturally large testes. Two to three days after injection the testes swell, the scrotum becomes reddened or purplish and glossy, adhering to the subjacent tissue, it being impossible to return the testes into the abdomen. The animal dies in 4-8-15 days. At autopsy one finds a violent inflammation of the tunica vaginalis, the serous membrane being covered by yellowish white granulations the size of a pin's head, the opposing surfaces being glued together by thick purulent exudate containing many glanders bacilli. The testicle and epididymis are very rarely affected. Usually this orchitis is sufficiently developed by the third or fourth day to make a diagnosis possible. The value of this test has been tried very generally (Straus, 1889; Hallipeau and Jeanselme, Finkelstein, Levy and Steinmetz, Kitt, and especially Nocard, 1891-96). At first its value was overrated. As Kitt pointed out, some guinea-pigs show a marked resistance, which takes away from the value of a negative result. The orchitis may be produced by other germs, *B. orchiticus* (Kutscher, 1896), which has the same morphological characters as *B. mallei*, but is distinguished therefrom by its retaining the Gram stain and behaving differently in cultures. The bacillus of epizootic lymphangitis (Nocard, 1892-96) is also different from *B. mallei* in these respects, consequently the staining test must also be applied to the organisms causing the orchitis. Secretion may be obtained from the nasal cavity by means of a sterile cotton swab, which is afterwards rinsed in sterile fluid for injection.

Mallein Test.—When mallein is injected into a glandered horse a typical reaction follows. After some hours an inflammatory swelling appears at the seat of inoculation; it becomes hot, tense, very painful, and always extensive, at times enormous. From the borders of the swelling hot and painful sinuous lymphatic swellings radiate toward the neighbouring glands. Suppuration never occurs where the inoculation has been carried out aseptically. The tumour increases in size for 24-36 hours, persists several days, subsides slowly, and disappears in 8-10 days. With the appearance of the swelling a general reaction takes place, the animal appears dejected, anxious, the coat ruffled and dull, breathing is accelerated, the appetite lessened. The animal shivers or has at times violent convulsive attacks. This constitutes the "reaction organique" of Nocard (1896), which is invariably accompanied by the febrile reaction which appears within 8 hours after the injection, and reaches its maximum in 10-12 or at times 15-18 hours. The rise of temperature amounts to 1·5°-2°-2·5° C., and persists for 24-48 hours. In healthy animals the injection of even much larger doses has little or no effect; a small, hot, œdematous and sensitive swelling may appear at the seat of inoculation, but it disappears entirely in less than 24 hours. A reaction has been observed in horses suffering from melanosis, bronchiectasis, and pulmonary emphysema, but it is not typical, and should be checked by the result of the Straus test. Where a doubtful reaction occurs the injection should be repeated after the temperature has returned to normal. To guard against error, animals should not be injected when showing fever, and they should be kept in the stable at a uniform temperature (changes in outside temperature have been observed to cause a variation of 1·5°-2° C. in the rectal temperature). (Kalning, Preusse, Pierson, Babes, Eber, Heyne, Schilling, Peters and Felisch, Dieckerhoff and Lothes, Bang, Nocard, Foth, Guinard, Teissier, Rabieux, Cadiot and Roger, Nikolsky, Schindelka, Johne, M'Fadyean, Remmert, Semmer, etc. etc., 1891-97).

Bonome (1894) tried the effect of mallein on a man suffering from chronic glanders of the nasal mucous membranes and cervical glands. A violent general reaction followed, the temperature rising after 4-7 hours. The mucous membrane of the nose and eyes became swollen, the pulse accelerated, the urine increased in amount. At first the reaction was proportionate to the amount of mallein used, but grew less with repeated doses; the patient, he says, showed marked improvement after two months' continued treatment.

X. Treatment.—The treatment of this disease is most unsatisfactory, and must be mainly symptomatic. If the case is diagnosed early, and an infective lesion found, it should be removed by the knife. The ulcers may be treated with antiseptics, and the fœtid smell kept within bounds by some deodorizer. In chronic cases fresh air, light, and good food will do more than any drug. Mallein has been claimed to be beneficial in the treatment of horses.

XI. Prevention.—As soon as it has been determined that glanders exists in a stable, all affected animals should be killed, and others which have been exposed should be isolated until they have been tested with mallein. The slaughtered animals should not be skinned, but put out of harm's way as soon as possible. Where the bodies cannot be rendered innocuous by boiling, destruction with chemicals, or fire, they should be buried at a spot some distance from the stable or house. The grave should be deep enough to allow of the cadaver being covered by a layer of soil at least 3 feet deep. As soon as a positive diagnosis has been arrived at it should, if possible, be determined how long the symptoms have existed, and if animals from other parts have been exposed to the infection. Where this is the case the animals should be inspected and watched. Persons who have to do with glandered or suspected animals should be thoroughly warned regarding the danger to which they are exposed. The infected stable should be closed to other horses and to every person outside of those directly concerned. Stablemen attending diseased animals should keep away from healthy ones. Suspicious animals should be examined every two weeks by a competent veterinarian, and though they may be worked as long as they show no symptoms, they should be kept apart from the unexposed, and watched for 3-6 months. The harness, etc. used by such animals should be reserved for their use alone. Where glanders has existed, the stables, harness, blankets, saddles, waggon-poles or shafts, buckets, drinking-troughs, mangers, etc., should be thoroughly disinfected. The stables should be cleaned out and aired, the walls and wood-work whitewashed. Sulphur may be burned (20 grams per cubic meter space) or chlorine evolved (add 10 grams of hydrochloric acid to 5 grams fresh chloride of lime for each cubic meter), and allowed to act for 8 hours, all apertures in the stable being sealed. Sulphur should not be used when chloride of lime is applied. Where the floor is bad, and the ground under it liable to have become soiled with the discharges of sick animals, the floor should be removed, as also the surface soil beneath, lime being freely scattered upon the spot. Where floors are good they may be washed with milk of lime or carbolic acid solution. Bits, chains, and metal articles may be sterilised over a fire, or scrubbed with boiling water and carbolic acid solution; articles having no especial value should be burned. Harness and leather articles may be washed with hot soda solution (50 grams soda to 10 litres water) or with hot soap-suds, being afterwards dried and rubbed with carbolised oil. Saddles, etc. which will not bear such treatment may be exposed to sulphur or formalin vapours. From this and what has been said with regard to the mode of spread of the disease, other precautionary measures will suggest themselves in particular cases. Naturally personal measures are indicated for those who are exposed, the hands, shoes, or soiled clothing being disinfected with carbolic acid, etc., when soiled. The possibility of a glandered horse being used for the production of an anti-diphtheritic serum has been suggested as a source of danger to man, but Bonhoff (1897) finds that the 0·5 per cent carbolic acid added to such serum kills off the bacilli in 2-4 hours.

LITERATURE.—The literature of glanders is so extensive that it seems inexpedient to append a bibliography. The reader is, however, referred to the following sources:—*The Catalogue of the Surgeon-General's Library* (Washington, D.C.), *The Index Medicus*, BAUMGARTEN's *Jahresbericht*, *Centralblatt für Bakteriologie*, FRIEDBERGER and FROHNER's *Lehrbuch d. speciellen Pathologie und Therapie der Hausthiere* (Stuttgart, 1896), vol. ii., SIMS WOODHEAD's article "Glanders" in Allbutt's *System of Medicine*. The dates given in conjunction with the names of the authors mentioned in the preceding pages make it possible for the reader to readily verify the references.

Glaucoma.

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DEFINITION AND NATURE.—The disorder of the eye known as glaucoma depends essentially on an excess of pressure in the chambers. In the healthy eye the fluid which fills the chambers keeps the tunics in a state of moderate tension, and thus preserves the form of the globe. Its pressure is equal to about 25 mm. of mercury; it varies somewhat with the force of the circulation, but only within narrow limits. Under certain circumstances the balance between the secretion and the excretion of this fluid is disturbed, an accumulation occurs in one or other chamber, the pressure rises, the tunics become more tense, the circulation of blood in the choroid and retina is embarrassed, and the function of the eye is seriously disturbed. This is the condition which we call glaucoma. The name referred originally to a greenish discoloration of the pupil, but this, though sometimes seen in the later stages, is no necessary part of the disorder. The name as now used denotes the peculiar morbid process which depends on pressure.

When glaucoma arises in an eye which appears to be otherwise healthy, we call it *primary;* when it occurs as a complication of some other disorder of the eye, *secondary*. Every eye which is suffering from an excess of internal pressure, however caused, is in a glaucomatous condition.

CAUSES AND PATHOLOGY.—The ciliary processes secrete a fluid which nourishes the vitreous body and lens, and fills the aqueous chamber. A very small part of this fluid passes slowly backward through the vitreous and leaves the eye in the region of the optic nerve; by far the larger portion passes forwards through the pupil and leaves the eye at the angle of the anterior chamber by filtering into Schlemm's canal and the minute veins connected with it. Glaucoma appears to depend in all cases upon some change which retards or prevents the escape of the intraocular fluid. In the large majority of cases the filtration angle is compressed or closed; in a few it remains widely open, but its permeability is diminished by inflammatory changes in the filtration area, or by blocking with blood, albuminous exudations, lens matter, or other abnormal contents of the aqueous chamber. These changes arise in various ways.

Causes of Primary Glaucoma.—In persons whose eyes are predisposed to the disease, an acute attack may be brought on by exposure to cold and damp, by fatigue, hunger, loss of sleep, or excessive mental effort, by anxiety or depressing emotion, by constipation, vomiting, or alcoholic excess, in short by any condition which disturbs the cerebral circulation and causes congestion of the eyes. Sometimes an attack begins during the course of a febrile disorder, such as pneumonia or influenza, or it may arise through a local cause, such as an injury of the head, a contusion of the eye without apparent lesion, or a slight abrasion or burn of the cornea. In certain eyes, where the anterior chamber is shallow and the filtration angle already narrow, the use of atropine or any other dilator of the pupil may light up a severe glaucoma, by thickening the base of the iris and thereby causing a complete blocking of the filtration angle; it is chiefly in the eyes of elderly people that this disaster is to be feared.

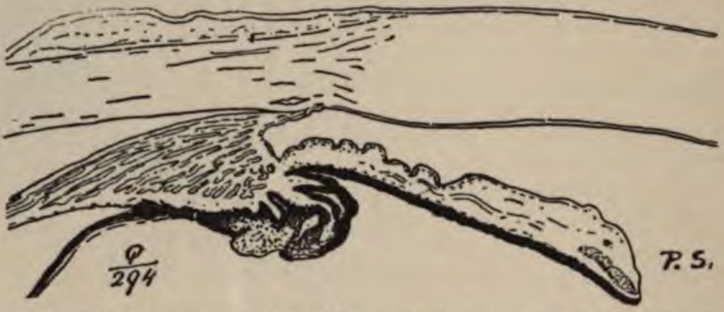

FIG. 1.—From the healthy emmetropic eye of a man aged 57. For comparison with Figs. 2 and 3.

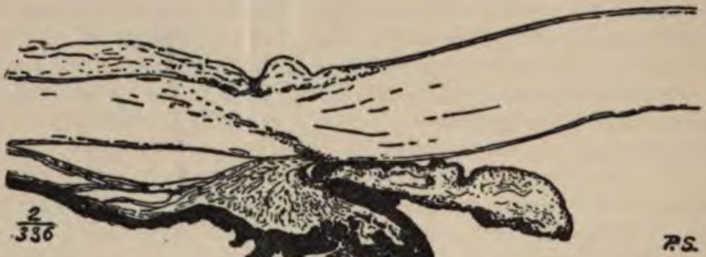

FIG. 2.—Primary glaucoma, acute, recent. Ciliary processes swollen and advanced; iris-base pressed against cornea, but not adherent to it.

If we examine an eye which has been recently blinded by acute glaucoma, we find the lens and ciliary processes pressed forward by an accumulation of fluid in the vitreous chamber; the processes much swollen and sometimes greatly altered in shape by pressure against the lens; and the base of the iris firmly pressed against the periphery of the cornea by the forward pressure of the processes. If the eye have been already blind for some months, as a result of acute glaucoma, the base of the iris will be found firmly adherent to the periphery of the cornea. At a later stage still the ciliary processes, instead of being enlarged and pressing forwards, will be found atrophied and retracted (see Figs. 1, 2, and 3). Swelling of the ciliary

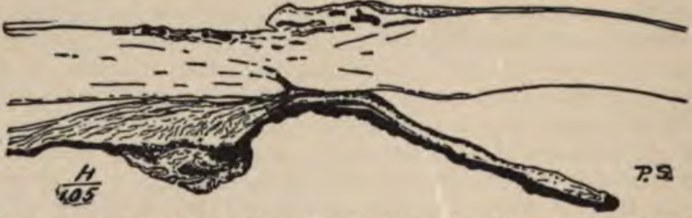

FIG. 3.—Primary glaucoma; chronic; duration about 12 months. Iris-base adherent to cornea; ciliary processes atrophied.

processes through venous congestion is probably in many cases the starting-point of the attack. In others there is probably some initial change in the zonula or hyaloid membrane, in the substance of the vitreous, or in the vitreous fluid itself, which prevents the overflow of this fluid from the vitreous into the aqueous chamber, and thereby leads to overfilling of the vitreous chamber. In other cases, again, it is probable that a forward displacement of the lens arises through loss of tension in the zonula. However it may be brought about, the compression of the filtration angle raises the pressure in the whole eyeball; this impedes the flow of blood through the choroidal veins and adds to the congestion and swelling of the processes; and this in turn aggravates the compression of the filtration angle. Acute glaucoma, like strangulated hernia, presents an obstruction of the circulation which intensifies itself, and which can only be relieved by removing the injurious pressure. In the slow chronic form of primary glaucoma there is little sign of vascular congestion, and there is no sudden or complete closure of the filtration angle; in the early stages the hindrance to the escape of the aqueous fluid is comparatively slight.

The predisposition to primary glaucoma appears to depend largely on certain structural changes or peculiarities in the eye which bring the lens into closer relations than usual with the parts around it. The crystalline lens, so long as it remains healthy, continues to increase in size throughout the whole of life, whereas the globe is fully grown at the beginning of adult life, if not earlier. Hence, as age advances, the lens encroaches more and more on the space in which it lies, its margin coming into closer relation with the ciliary processes, and its anterior surface approaching the cornea. In some eyes these changes pass the limit of safety; the ciliary processes, having insufficient free space at their disposal, are apt, especially during periods of turgescence, to press against the iris-base, and thus compress the filtration angle, and the iris being already very near to the cornea, tends, when the pupil is dilated, to block the outlet. In accordance with these natural changes in the eye, the liability to primary glaucoma steadily increases throughout life; it is extremely slight in childhood and early adult life; it increases in each decade; it is manifest chiefly in elderly people; at sixty-five years of age, for example, the liability to the disease is at least twice as great as at forty-five. The liability of women is greater than that of men in a ratio probably of about six to five, and in women the disease more often occurs in the acute congestive form.

Again, in eyes of small size the relations of the lens are often faulty. The growth of the lens appears to be little influenced by variations in the size of the parts around it, and in eyes in which the cornea and sclera have failed to reach their full development the lens is apt to be relatively too large. This is very obvious in certain cases of microphthalmos. A small eye may usually be recognised during life by smallness of the cornea. The horizontal diameter of the cornea measures normally from 11 to 12 mm.; among eyes in which it measures less than 11 mm. we find a higher percentage of glaucoma, and those in which it measures as little as 10 mm. seem seldom to escape the disease.

The predisposition to glaucoma is sometimes distinctly hereditary. In one such case observed by the writer both parent and child had exceptionally small eyes.

Certain races—Jews, Egyptians, negroes—are said to be specially liable to the disease, but on this point more precise evidence is wanting. Among Egyptians the average cornea is said to be smaller than among Europeans, and smaller in eyes suffering from primary glaucoma than in healthy eyes.

Hypermetropia probably predisposes somewhat to glaucoma in advanced life, for the hypermetropic eye has as a rule a more prominent ciliary body and perhaps a shallower anterior chamber than the emmetropic or myopic eye, and these conditions tend to facilitate compression of the filtration angle. But the influence of hypermetropia in this respect has hardly yet been proved by statistics; it must be remembered that the acquired hypermetropia produced by changes in the lens in advanced life is a very common condition at the age when glaucoma most commonly begins, and it must not be assumed that small eyes are necessarily hypermetropic, for this is not the case. It is the small eye as such rather than the hypermetropic eye that is peculiarly liable to primary glaucoma.

Apart from these physiological variations it is probable that degenerative changes in the blood-vessels and other tissues of the eye account in part for the predisposition to primary glaucoma which belongs to advanced life.

Causes of Secondary Glaucoma.—Sarcoma of the choroid usually leads to glaucoma unless the eye be excised before the outbreak. The glaucoma is of acute type and closely resembles the primary form of the disease. The growth of the tumour is accompanied by serous effusion from the choroid; the retina is detached and driven inwards; at first the vitreous makes room for the intrusion by parting with some of its fluid through the hyaloid into the aqueous chamber and so out of the eye; later, when the vitreous fluid is nearly all gone, further compensation is impossible; the lens, ciliary processes, and iris are driven forward, and the filtration angle is compressed just as in primary glaucoma. Glioma of the retina commonly leads in like manner to high tension, but the attack is of less violent character. Tumours of the iris may induce glaucoma by direct blocking of the filtration angle.

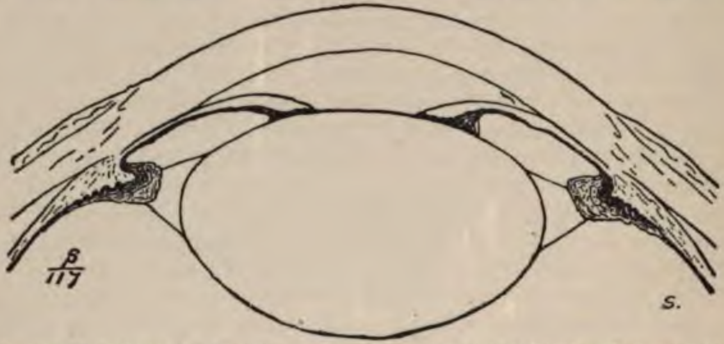

Fig. 4.—From an eye blinded by secondary glaucoma following neglected iritis. Exclusion of the pupil; accumulation of fluid in the posterior chamber; displacement of the iris; closure of the filtration angle.

Intraocular hæmorrhage is sometimes the starting-point of a condition closely resembling primary glaucoma. It is important to distinguish these cases when possible, for hæmorrhagic glaucoma is less amenable to treatment.

Annular posterior synechia leads to secondary glaucoma by preventing the passage of the aqueous fluid forwards through the pupil. The iris is bulged forward by the accumulation of fluid behind it, the periphery of the chamber is abolished, and the eye is quickly lost by high tension unless an aperture be made in the iris by operation (see Fig. 4).

Perforating wounds and ulcers of the cornea lead to secondary glaucoma in certain cases through partial or total abolition of the anterior chamber, the iris being applied to the cornea. So long as the wound leaks, the eye remains soft; when leakage ceases, it becomes hard. The danger of this complication is increased when the injury is complicated by wounding of the lens.

Dislocation of the lens into the anterior chamber is sometimes followed by intense glaucoma. In such cases we can see that the iris is closely applied to the posterior surface of the lens and to the periphery of the cornea by the accumulation of fluid behind it. Lateral dislocation of the lens, as caused by a blow on the eye, is apt to lead to closure of the filtration angle by the pressure of the lens on the one side of the circle, and by the pressure of the displaced vitreous at the other.

Operations for the removal of cataract, whether by extraction or needling, are occasionally followed by secondary glaucoma. In all such cases careful examination shows compression of the filtration angle either by the swollen lens, or by the traction of membranes stretched across the pupil, incarcerated capsule, entangled iris or vitreous, or in some such way.

Cyclitis in certain instances leads to secondary glaucoma, the ciliary body pouring out a morbid albuminous fluid which, unlike the normal aqueous, is unable to permeate the filtration angle and therefore tends to accumulate in the anterior chamber, which is deepened thereby. In this and in some other allied forms of the disease the high tension is due not to narrowing of the outlet, but to abnormal constitution of the fluid.

The rare form of glaucoma sometimes present at birth has been found to depend on congenital absence of the filtration angle, the condition being probably a fault of development rather than a product of disease. The glaucoma occasionally met with in eyes which present a partial or total absence of the iris has been found to depend on blocking of the filtration area by a rudimentary structure, not visible in the living eye, which represents the iris.

It is obvious from the foregoing that the remote causes of glaucoma are very various, and include constitutional diseases; disorders of the respiratory, vascular, and nervous systems; injuries, morbid growths, congenital imperfections; and senile changes. The immediate cause of the high tension appears to be in all cases an obstruction which leads to accumulation of fluid within the eye.

Symptoms.—The symptoms of glaucoma are numerous and complex, and vary considerably in different forms of the disease. They are the results of the pressure within the eye. In primary glaucoma we see these pressure changes in their uncomplicated form; in secondary glaucoma they are more or less modified by the presence of other changes in the eye. Before describing the clinical varieties of primary glaucoma it will be well to consider the various pressure symptoms from the point of view of their causation.

Increased tension of the eyeball is the leading symptom of glaucoma. Every medical man should know how to detect it:—The patient is told to look downwards; the surgeon, standing before him, steadies his hands by resting the outer fingers on the forehead, places the tips of his two forefingers on the upper eyelid, and feels the globe behind the margin of the cornea with gentle alternating pressure. He then feels the fellow-eye in like manner for comparison. It must be remembered that prominence of the eyeball is no evidence of increased tension, and that persons whose tension is normal frequently complain of a sense of fulness in the eyes. The following symbols are employed to describe variations of tension:—

TN: Tension normal.
T + 1 ?: Doubtful increase of tension.
T + 1: Slight but positive increase of tension.
T + 2: Considerable tension; the finger can slightly impress the coats.

T + 3 : Extreme tension ; the finger cannot dimple the eye by firm pressure.
T – 1 ? : Doubtful reduction of tension.
T – 1 : Slight but positive reduction of tension.
T – 2, T – 3 : Successive degrees of reduced tension.

Many attempts have been made with more or less success to replace the finger test by an instrument of precision—the tonometer. The instrument designed and used by the writer is so constructed as to make a known pressure on the surface of the sclera and to indicate the depth of the impression produced. For estimating doubtful changes of pressure it is a useful aid in many cases, but in daily work no instrument can replace the finger test.

Injection of the Eye.—A sudden access of high pressure, by embarrassing the internal circulation, and by reflex nerve action, causes intense engorgement of the external vessels, with more or less œdema of the conjunctiva, swelling of the lids, and sometimes even proptosis. In less violent attacks the visible injection is limited to the ciliary zone and the larger vessels which pass backwards from it. In glaucoma of gradual onset there is little or no visible injection of the eye beyond some enlargement of the main anterior ciliary vessels, arteries as well as veins. The ciliary arteries especially are more prominent and more difficult to empty by finger pressure than in health.

Pain.—Pain varies with the amount of vascular disturbance. In acute attacks it is sometimes extremely severe and radiates through all the branches of the fifth nerve. In slow non-congestive glaucoma it is often entirely absent, or occurs only in the last stage when congestion supervenes.

Cloudiness of the Cornea.—The lymph streams which traverse the normal cornea are hindered in their flow when the tension of the tunics is suddenly raised. A condition of slight œdema is set up, minute drops of fluid collecting beneath the anterior epithelium and between the more superficial fibres. It is this œdema of the cornea in its incipient stage that causes the appearance of rainbows round the light which many glaucoma patients notice at the beginning of an attack. When more pronounced it causes a visible cloudiness of the cornea and is in large part the cause of the extreme obscurity of vision which occurs during the height of an acute attack. The glaucomatous opacity of the cornea is distinguished from all forms of inflammatory opacity by the rapidity with which it appears and disappears in connection with changes of pressure in the eye. When high pressure is long continued the corneal epithelium may thicken and separate in vesicles or blebs ; the opacity is then denser and more permanent. In glaucoma of very gradual onset the œdema of the cornea is entirely absent until the later stages of the disease are reached.

Anæsthesia of the Cornea. — During acute attacks, and in the later degenerative stages, the cornea partly loses its sensibility, probably through maceration and compression of the nerve filaments by the fluid collected beneath the epithelium, and their rupture when the epithelium is separated.

Dilatation of the Pupil.—In acute glaucoma the pupil is dilated. This depends probably on a lowering of the blood supply to the iris produced by the rise of pressure and aggravated by the compression of the iris-base between the turgid ciliary processes and the cornea. The compression affects the nerves of the iris as well as the vessels, and the dilatation ultimately becomes permanent through paralysis and atrophy of the sphincter. The oval dilatation seen in some cases probably shows that the compression has been more severe at some parts of the circle than at others. In chronic glaucoma, on the other hand, the vessels of the iris have time for a compensatory hypertrophy, and the blood-supply is maintained in spite of the gradually rising pressure in the eye; there is, moreover, little if any pinching of the iris base. In these cases, accordingly, the pupil dilates but little and sometimes not at all. It may remain of normal size and consensually active, even after the eye is blind. In those forms of secondary glaucoma which are associated with posterior synechia, there is of course no dilatation of the pupil.

Loss of Accommodation.—The range of accommodation is usually diminished by the onset of glaucoma, probably because the pressure in the eye makes the choroid more tense and increases its resistance to the contraction of the ciliary muscle. In the later stages the muscle atrophies.

Changes in the depth of the Anterior Chamber.—Shallowing of the anterior chamber is a common symptom in primary glaucoma, and in the allied forms which are induced by choroidal tumours and retinal hæmorrhage. Deepening of the chamber is characteristic of the secondary glaucoma of serous cyclitis and of congenital buphthalmos. In cases of the former type the lens is pushed forward by accumulation of fluid in the vitreous chamber; in the latter it is pushed backward by retention in the aqueous chamber.

Changes of Refraction.—Increase of pressure may influence the refraction in several ways. Supposing the eye to be naturally emmetropic, forward displacement of the lens induces myopia, backward displacement hypermetropia. The elongation of the globe which occurs in the glaucoma of children induces myopia of high degree. In the majority of cases the refraction, if altered at all, is increased. It is often found higher during a glaucomatous attack than before or after. Moderate pressure long continued appears in some cases to induce astigmatism of exceptional kind, the horizontal meridian of the cornea becoming more sharply curved than the vertical. The opposite condition, commonly found after an upward iridectomy, is of course due to a flattening of the vertical meridian induced by the marginal incision.

Excavation of the Optic Disc.—Under continued pressure the optic papilla is transformed into a cup. The lamina cribrosa,—the sieve-like part of the sclera which gives passage to the optic nerve fibres,—being the weakest spot in the wall of the eye, is displaced backward, together with the nerve-fibres and blood-vessels which it supports. The firmer ring of sclera around it limits the area of excavation. The nerve-fibres, being bent and stretched over the unyielding margin, suffer atrophy and loss of bulk, and a deep undermined cup results. Cupping is not found during or after a first attack of acute glaucoma, for the atrophy takes time. On examining a cupped disc with the ophthalmoscope by the indirect method, and making small lateral movements of the object lens, we see that the vessels in the plane of the retina have a greater apparent movement than those at the bottom of the cup, and seem to outrun and travel in front of them. By the direct method we see the difference of refraction between the margin and the bottom of the cup, and the depth of the latter may be roughly estimated by remembering that a difference of three dioptres corresponds to a difference of level of about 1 mm. The floor of the cup is paler than the normal disc and more distinctly cribriform, through atrophy of the nerve-fibres. The sides are more or less hidden by the overhanging margin, so that the vessels seen on the floor are lost to view as they ascend the sides, and reappear changed in number and position as they bend round the margin to gain the retina. Around the margin there is usually some atrophy of the choroid visible as a zone of lighter colour than the adjacent fundus. The vessels are more or less displaced toward the nasal side of the cup. When associated with posterior staphyloma, as in high myopia, the glaucoma cup is larger in diameter than usual; its sides are little if at all undermined; its vessels are attenuated by elongation and comparatively free from sharp bends or curves. The glaucoma cup is to be distinguished from the so-called physiological cup—a central depression often present in the healthy disc—by the fact that it involves the whole area of the disc. From the shallow excavation of simple atrophy it is to be distinguished by its depth and by the sudden bending and interruption of the vessels at its margin. These distinctions, however, are not always easy to make, and when simple atrophy occurs in a disc in which there is already a large physiological cup, the resulting condition may closely resemble the typical glaucoma cup.

Pulsation of Retinal Vessels.—The pressure on the retina obstructs the entrance of the arterial and the exit of the venous streams. Hence the arteries are apt to be incompletely filled, the veins congested. Arterial pulsation, rarely visible in a healthy eye, is often to be seen in the area of the disc, or may be induced by light finger pressure on the globe. The veins are rhythmically compressed during each arterial pulsation by the pressure transmitted to them through the vitreous. This venous pulsation occurs only in a small portion of the vein close to its point of exit, for the blood-pressure is lowest here, and compression at this point prevents the expulsion of the blood from the adjacent part of the vessel. It is often visible in eyes of normal tension, but not so often as in glaucoma. Capillary hæmorrhage may result from the obstruction of the retinal circulation. Aneurysmal dilatations of the arteries and beadlike varicosities of the veins are occasionally seen.

Impairment of Vision.—Vision is affected in several ways by the pressure in the eye.

The transient œdema of the cornea which occurs when glaucoma is threatening causes temporary obscuration of vision in the daytime, and an appearance of a ring of rainbow colours around every luminous flame at night. The characters of this rainbow vision are these: The flame is seen with nearly normal clearness; around it is a dark non-luminous zone, the breadth of which on each side of the flame corresponds to an angle of 4° to 5°. Surrounding this is the coloured zone, which has an angular breadth of 2° to 2°·5, and a total diameter of about 10° to 11°. In the coloured zone the whole of the colours of the spectrum are visible, the violet being invariably at the inner, the red at the outer border. The zone is not altered either as to size or as to position of the colours by the use of convex or concave lenses, and it is not altered by variations in the diameter of the pupil. It is perceived not only in direct vision, but also, though with less distinctness, when the image of the flame falls on other parts of the retina than the yellow spot. An appearance corresponding in all respects with the rainbow vision of glaucoma can be induced experimentally by the application of a single drop of a 0·125 P.C. solution of hydrochlorate of erythrophleine to the eye; at the same time a faint haze of the corneal epithelium occurs. This experiment, while it clearly shows the dependence of rainbow vision on a disturbance of the corneal epithelium, proves also that it is not necessarily dependent on an excess of pressure in the eye. The much-dreaded rainbows, therefore, are not invariably a sign that glaucoma is approaching. They occur also in some congestive states in which no increase of tension is discoverable.

Further, the sensibility of the retina is lowered when an excess of pressure falls upon it, as any one may discover for himself by making finger pressure on the eyeball—an experiment not to be made frequently or incautiously. Sensibility may be abolished by pressure, over the whole area of the retina, but less easily in the region of the macula than elsewhere. Acute glaucoma is attended with a more or less complete retinal paralysis of this character. It appears to depend on a disturbance of the circulation in the retinal vessels, and perhaps in the choroidal plexus, on which the rods and cones of the retina depend for their nutrition. Chronic glaucoma is attended by retinal paralysis of a different kind. The field of vision fails at the periphery, while at the centre it remains for a while intact. This peripheral contraction of the field is discoverable at first only in a subdued light. It commonly begins at the inner or nasal margin of the field, involves the inner, upper, and lower portions before the outer, and slowly advances till it has reduced the sentient area to a small oval or slit-like form extending outwards from the fixation point to the blind spot and beyond it. It next involves the fixation point and the adjacent area, so as to leave only a small eccentric area of vision extending outward from the blind spot, corresponding to a small area of the retina extending inwards from the optic disc. Ultimately this fails also and the eye is blind. This contraction of the field is probably the expression of progressive damage of nerve-fibres in the excavated disc, those belonging to the temporal half of the retina suffering earlier than those belonging to the nasal half; those passing to the periphery earlier than those supplying the macular region. The contraction does not always follow the typical course here described, but the comparative immunity of the central and outer parts of the field during the failure of the remainder is found in a very large majority of cases. This characteristic, together with the relatively sharp line of demarcation between the sentient and non-sentient areas, and the relatively good retention of colour vision in the former, help to distinguish the contracted field of chronic glaucoma from that of primary atrophy of the optic nerve. When the field is tested by means of very minute test objects defects are found which would otherwise escape detection, and these defective areas, wherever situated, appear to be in direct continuity with the blind spot of the optic disc. The two types of retinal paralysis are often combined. An acute glaucoma initiates the loss of vision by obstructing the circulation; prolonged pressure completes it by excavating the disc. Timely removal of the pressure will do much to restore what is lost through recent interference with the blood-supply, but the loss due to atrophy of nerve-fibres is permanent.

CLINICAL VARIETIES OF PRIMARY GLAUCOMA.—*Acute primary glaucoma* is apt to be mistaken for an inflammation of the eye, an attack of erysipelas, or a neuralgia. In some cases it arises without known cause and without warning of any kind. More frequently it is preceded by one or more transient attacks of dimness and rainbow vision, and is attributable to chill, exhaustion, loss of sleep, or some other of the exciting causes which have already been discussed. In some cases it shortly follows the use of atropine or some other dilator of the pupil. Pain begins in the eye more or less suddenly, increases from hour to hour, and extends over the side of the head and face. In the severest cases vomiting occurs and the patient is much prostrated. Vision is much impaired. When summoned to the case the surgeon probably finds the conjunctiva much injected, and the eyelids, in severe cases, somewhat swollen. The subconjunctival vessels are engorged, especially as regards the circum-corneal zone and the main trunks connected with it. The cornea is more or less wanting in polish, and if high tension has already persisted some days its sensitiveness to touch is diminished. The pupil is more or less dilated, totally inactive, and sometimes somewhat oval; the dilatation in conjunction with the injection of the eye should at once arouse a suspicion of glaucoma. The anterior chamber is usually shallow. The eyeball is decidedly too hard under the finger test, and this fact establishes the diagnosis. The vision of the eye may in the course of a few days, or even sooner, be reduced to a mere perception of light near to the middle of the field, or it may be totally extinguished. Ophthalmoscopic examination is usually prevented by the cloudiness of the cornea; but, if visible, the retinal arteries will be seen to be somewhat reduced in size and perhaps pulsating, the veins engorged, and, at their point of emergence from the disc, collapsing with each arterial pulse. Hæmorrhages are sometimes discoverable in the retina. Such an acute attack, unless relieved by treatment, will commonly last for several weeks and then slowly subside, but though the more prominent symptoms disappear, the eye remains hard and the remnant of sight is gradually lost. The most acute cases have been termed fulminating from the violence of their onset; the less acute form a connecting link with the subacute variety. The more acute the attack the greater the need for prompt and efficient treatment if sight is to be saved.

Subacute primary glaucoma resembles the acute form, but is milder and for a while intermittent. At first there may be merely temporary periods of mistiness and rainbow vision. Sooner or later the recurrences become more frequent, the intermissions less complete, and a persistent congestive glaucomatous condition is established. Blindness comes on less rapidly than during acute glaucoma, but not less surely. These congestive forms of the disease are spoken of by many writers as inflammatory glaucoma, and it is true that inflammation attends their course; but, seeing that the condition is essentially one of mechanical obstruction, and not of primary inflammation, the term is apt to mislead, and is on that account objectionable.

Chronic primary glaucoma (simple glaucoma) is apt to pass unnoticed even by the patient in its first stage, and may be mistaken by the medical man for simple atrophy of the optic nerve. It begins almost imperceptibly, progresses slowly with little tendency to exacerbation or remission, and leads, in the course of months or years, unless arrested by treatment, to total blindness. The patient is usually at least fifty years of age. He complains of failing sight in one eye or in both. One eye is usually affected first, the other almost always follows sooner or later. There is usually no history of periodic obscuration, and the onset of the disorder cannot be exactly dated. It sometimes appears to have been preceded by a period of mental strain of some kind. Externally, the eyes exhibit little or nothing amiss. The chief anterior ciliary vessels may be somewhat enlarged, the anterior chamber rather shallow. One or both pupils may be somewhat dilated, but this is not a constant symptom. An eye already blind through chronic glaucoma may have a pupil which is equal to that of the seeing eye, and which acts consensually with it. The lens, on simple inspection, may appear to be more or less cataractous, and yet, when examined with the ophthalmoscope, prove to be quite transparent. Mistakes of diagnosis from this cause are not infrequent and are very unfortunate, for a delay which may be wise in the case of cataract is disastrous in the presence of glaucoma. The diagnosis depends chiefly on the tension of the eye, the field of vision, and the condition of the disc. The tension is increased, but in some cases, especially in the earlier stage, the increase may be very slight and at times absent. When possible, it should be tested at different times of day. The same eye may be quite normal to the touch at one time, distinctly tense at another. It is usually towards night, when the patient is fatigued, that the increase is most perceptible. In the later stages the eye may become extremely hard, and yet, by reason of the slow progress of the disorder, there may be no pain and little external sign of disease. The field of vision will show, when mapped by means of the perimeter, the characteristic and gradually increasing contraction which has been described. The optic disc will show the typical glaucoma cup. The course of the disease is slow, and in exceptional cases may even extend over many years before the stage of complete blindness is reached.

Absolute Glaucoma.—When the eye has lost perception of light in all parts of the field the glaucoma is said to be absolute, and in this stage the ultimate effects of high pressure are gradually developed. If the glaucoma have been acute or subacute, the disturbances of the eye remain very obvious. The external vessels are large and tortuous, and stand out prominently in the atrophied conjunctiva; the cornea is hazy, its epithelium thickened, and its sensibility lowered or abolished; the iris, if visible, is seen as a narrow circle by reason of the wide dilatation of the pupil, and it may be visibly in contact with the cornea; enlarged blood-vessels may be visible on its surface; the lens degenerates and ultimately becomes opaque, sometimes conspicuously white or yellow. Pain is apt to continue or to recur from time to time, and it is for this that the patient seeks relief. Subjective visual sensations may delude the patient into hoping for some recovery of sight. If the glaucoma have been chronic throughout, without pain or congestion, the appearance of the eye may be nearly normal even in the absolute stage, but the high tension, and the excavation of the disc, at once reveal the cause of the blindness. Sooner or later, however, such an eye is apt to suffer attacks of pain and congestion which demand relief, and this relief can be given more speedily and certainly by removal of the eye than in any other way. The last stage of glaucoma is marked by gradual thinning and extension of the wall of the eye, commonly in the region of the equator, sometimes in the ciliary zone. Rupture of the globe occasionally completes the destructive process.

Secondary Glaucoma.—The varieties of secondary glaucoma need not be further described. The diseases of the eye which are liable to this complication have been enumerated. Increase of tension is the essential sign of its onset. Vascular injection, pain, and impairment of vision, if already present, are aggravated by the access of high pressure in the eye. The pupil, if it be free, dilates. The field of vision, if not already lost, contracts. The optic disc in course of time suffers excavation, though this latter change is commonly hidden from observation in the living eye, and discoverable only after the eye has been removed.

TREATMENT.—The successful treatment of glaucoma depends essentially upon lowering the increased pressure within the eye, and the measures by which this can be effected must be employed in the earlier stages of the disease if permanent loss of sight is to be avoided. When the eye is blind, treatment can avail nothing beyond the relief of pain or the removal of disfigurement, and in most cases these ends are attainable more certainly, speedily, and safely by complete removal of the useless organ than in any other way.

Glaucoma usually calls for operative treatment, for it is seldom possible, otherwise than by operation, to re-establish a permanent and sufficient outlet for the intra-ocular fluid. There are many cases, however, in which certain auxiliary lines of treatment are of great value, and there are a few in which benefit of long duration may be obtained by these means alone. It is chiefly primary glaucoma which we have here to consider.

Myotics.—Drugs which contract the pupil, of which those chiefly used are physostigmine (eserine) and pilocarpine, sometimes rapidly relieve the high tension. They are the antagonists of atropine, which dilates the pupil and, where predisposing conditions exist, induces or aggravates high tension. It is to be noted that neither myotics nor mydriatics cause any decided change of tension in normal eyes. Their action in relation to glaucoma appears to depend mainly on the abnormal position of the iris. Eserine and pilocarpine, by contracting the sphincter of the pupil, attenuate the iris, flatten out its folds, and pull upon its peripheral insertion. These changes tend to reopen the filtration angle when it is narrowed or compressed. Accordingly, it is chiefly in the comparatively mild attacks which come and go in the early stage of the disease that we can rely upon their action. In severe acute attacks they may be very useful if used without delay, but in these cases the iris base is firmly compressed, it forms adhesions early, and the sphincter of the pupil soon loses its contractility; hence the stage in which myotics can relieve these violent attacks is soon at an end. In chronic non-congestive glaucoma some lowering of tension may be obtainable over a considerable period of time by a continued use of eserine or pilocarpine, but the improvement is seldom great or permanent. Of these two drugs, if used in equal quantity, eserine is much the more active and the more irritating; a one-half per cent solution of sulphate of eserine in distilled water (roughly two grains to one ounce) is amply strong enough for any purpose, and in most cases a much weaker solution is better. A one or two per cent solution of nitrate or hydrochlorate of pilocarpine appears in many cases to be equally effective and to be better tolerated. These drugs should be used in the minimum amount, and with the minimum frequency which suffice to contract the pupil and to keep it contracted: usually one drop once or twice a day, sometimes more frequently, and in some cases of chronic glaucoma, at longer intervals. In cases where slight temporary attacks occur at considerable intervals of time, the drops should be used only as often as is necessary to banish them. In cases in which the contractility of the pupil is entirely lost myotics are probably useless, and in these same cases atropine is probably harmless, and sometimes perhaps beneficial. When atropine cannot dilate the pupil it never probably raises the tension, and in those forms of secondary glaucoma characterised by serous exudation into the aqueous chamber, with deepening of the anterior chamber, atropine tends to lessen the inflammation and thereby to restore the normal tension. Apart from their influence on the iris and ciliary muscle, however, the action of these drugs is not yet completely known, and in some cases of glaucoma, especially during treatment after operation, and in some forms of secondary glaucoma, the choice between a myotic and a mydriatic is not easy to make; the one or the other must be used tentatively, and its action carefully watched.

Cocaine, like every other dilator of the pupil, may, under predisposing conditions, induce or aggravate glaucoma. It has, however, the power of contracting the ciliary blood-vessels and diminishing the sensibility of the ciliary nerves—effects which tend to lower the intra-ocular pressure. It is often useful, therefore, to combine cocaine with eserine or pilocarpine in such proportions that, while the cocaine helps to subdue the vascular congestion, the myotic keeps the pupil contracted.

Morphine given subcutaneously, or by the mouth, will sometimes rapidly cut short an acute attack, and is useful in many cases. It eases pain, promotes sleep and contraction of the pupil, lowers the blood-pressure, and probably diminishes for a time the secretion from the ciliary processes. *Antipyrin* and some other drugs of the same class are useful in the same way; they can be repeated with more freedom than morphine, but have a less positive influence over the tension of the eye.

Sleep, even though of very short duration, and occurring without the aid of any drug, often dispels the mild premonitory attacks by which primary glaucoma is ushered in. During sleep the pressure in the cerebral vessels falls and the pupil contracts. *Warmth, food*, and *rest* relieve, just as cold, hunger, and fatigue induce these early and slight attacks.

Aperients are often needed, and sometimes have a well-marked effect in lessening the fulness of the cerebral vessels and the pressure in the eye. The habit of straining at stool should be expressly forbidden; it congests the head and tends to aggravate the glaucomatous condition.

Ice applied over the closed lids, in the form of ice compresses or in a thin rubber balloon, is sometimes useful in conjunction with other measures in presence of acute congestion.

By a judicious combination of these palliative measures, especially by contracting the pupil, relieving pain, and inducing sleep, we can frequently, especially in early acute cases, obtain great improvement for a time, and thereby postpone the time at which an operation is imperative; we can then operate under more favourable conditions; but the temporary benefit obtained in this way is likely to do more harm than good if it leads to undue delay of operative treatment.

Operative Treatment.—The object with which we operate is to obtain a permanent and sufficient outlet for the intra-ocular fluid. The most trustworthy operation in the large majority of cases is *iridectomy*. The improvement of vision obtainable by iridectomy varies with the type of the disease: it is greatest in the acute form, smallest in the chronic. An acute attack may in the course of a few days reduce vision from its normal condition to a bare perception of light; in such a case an iridectomy promptly performed may restore it nearly, if not quite, to its former condition. A chronic glaucoma, on the other hand, may advance slowly and insidiously for a year or more, excavating the disc and contracting the visual ~~fluid~~ field, but causing little change in the acuteness of central vision as indicated by test types. An iridectomy in such a case may enlarge the visual field a little; it may arrest the progress of the malady; it may preserve the vision which still remains but cannot greatly improve it. The urgency for operation varies, of course, with the acuteness of the disease. In acute glaucoma, unless improvement is rapidly obtained by the measures already discussed, iridectomy should be performed without delay. The prostration of the patient, though severe, is no reason for delay, for an iridectomy is the surest means of giving ease and sleep as well as of saving the eyesight. If light-perception be already completely lost we cannot hope for restoration of good vision, but if it has not been lost many days, it is still right to operate at once, for there is still the chance of recovering some amount of vision. In subacute glaucoma also it is important to operate early, for each recurrence of the acute symptoms is likely to render a perfect operation more difficult of attainment, and a perfect recovery of vision less probable. In such cases it is best to cut short the acuter symptoms if possible by palliative treatment and to operate during a quiescent period. In chronic non-congestive glaucoma the benefit obtainable by iridectomy is less obvious and less certain; positive improvement of vision is seldom to be hoped for; the object of the operation is to prevent further loss. Even this is not always achieved. The operation sometimes fails to arrest the course of the disease, and occasionally accelerates it. When repeated examinations of the eye at intervals of a week or two, or longer, have shown that the visual field continues to contract, and that the tension remains in excess, in spite of a careful trial of myotics, then it is usually our duty to advise operation. It is the only means by which the eye can be saved from certain blindness, but it is not a certain means, and this should be carefully explained to the patient, or in some cases to his friends, before it is undertaken. If both eyes are affected, but both still retain some sight, the worse should be operated on first. The result may decide the treatment of the other. Some surgeons advise their patients, especially if there be already great contraction of the field, to accept the gradual approach of blindness rather than the risk of operation, and in some cases, where the probable duration of life is short, this is certainly the right course, but as a general rule our duty is to advise operation so long as the eye retains sight worth saving. Cases of definite arrest of chronic glaucoma, with retention of useful vision over many years, are known to all experienced operators. The benefit obtained must be estimated afterwards rather by the field of vision which is retained than by the central acuteness—that is, rather by the perimeter than by the test types.

Mode of Performing Iridectomy.—The operation consists in making an incision which opens the anterior chamber near to its periphery, and removing as completely as possible the corresponding portion of the iris. When the eye is painful a general anæsthetic is usually required; in chronic non-congestive glaucoma cocaine will usually suffice, and we have then the advantage that we can control the position of the eye by letting the patient look at a lighted candle held in the requisite position. The incision is made with a small linear cataract knife. It is usually placed at the upper part of the cornea. The puncture is in the sclera at least 1 millimetre from the corneal margin; the counter-puncture is at a corresponding point; the distance between puncture and counter-puncture externally in a straight line is from 7 to 9 millimetres, according to the size of the cornea and the depth of the anterior chamber. When the iris lies very close to the cornea it is impossible to make a full-sized incision. In passing across the chamber the knife is kept parallel with the plane of the iris; in cutting out the edge is turned somewhat forward, but must come out well in the sclera and raise a conjunctival flat. The iris is seized with the small forceps, drawn out through the incision, and lightly pulled to the right and to the left, so that its base may be disengaged from the cornea. It is then divided with scissors close to one end of the incision, drawn towards the other end, and if possible torn at its root; is again drawn away from the angle of the wound so that it may not be pinched and incarcerated; and is removed with a second snip of the scissors. Some operators make the incision with a broad lance knife instead of the cataract knife. In the writer's opinion, the cataract knife enables the surgeon to make a more peripheral incision than can be safely made with the lance, and to modify its length and position, according to the space available, after the point has appeared in the chamber, by making the counter-puncture a little farther forward or backward as may be found practicable. Some operators place the incision at the part of the circle where the iris appears to respond most readily to myotics, on the ground that removal of the iris-base is more likely to be attained here than elsewhere. For a laterally placed incision the lance knife must be used. The upward incision has the advantage that it can be made with the linear knife, and that the coloboma lies under the upper lid. In the writer's opinion, it is advisable in most cases to slacken the eye by a scleral puncture immediately before making the iridectomy. (See later.)

A successful iridectomy permanently removes the excess of tension either by causing the normal filtration outlet to reopen or by establishing an abnormal one. In a recent acute glaucoma a good iridectomy permits an escape of blood from the turgid ciliary vessels, and an escape of clear fluid from aqueous and vitreous chambers. The ciliary processes recede and cease to compress the base of the iris, and with the re-establishment of the anterior chamber the filtration angle reopens: the normal outlet resumes its function. This has been proved by the examination of eyes cured years previously by iridectomy. The absence of the iris segment appears to act as a safeguard against the recurrence of a similar blockade. In congestive glaucoma of longer standing restitution of the filtration angle is often unattainable, but even in such cases a well-made iridectomy will sometimes free the ligamentum pectinatum in the region of the wound by tearing away the iris from its root. In many of these cases, however, and in chronic glaucoma also, the success of an iridectomy depends on the formation of an artificial filtration channel: a minute, permanent, corneo-scleral fistula. The lips of the wound, especially the inner lips, do not unite completely, but remain more or less separated by the prolapse between them of a fold or tag of iris. The aqueous continues to escape at this point into the subconjunctival tissue, and is thence absorbed. The overlying conjunctiva presents an œdematous or pearly appearance, and is more or less elevated by the collection of fluid beneath it. Finger pressure carefully applied to the eye day after day during the healing process aids in keeping the tension low, and probably promotes the formation of such fistulæ. Even weeks after an iridectomy firm pressure with the finger will sometimes cause an immediate visible extrusion of fluid beneath the conjunctiva, with slackening of the globe. In chronic glaucoma, and in the later stages of the congestive forms, a slightly fistulous scar affords the best, if not the only, guarantee against a speedy return of the glaucoma, and is therefore a result to be desired. After an iridectomy for glaucoma there is usually a considerable flattening of the cornea in the meridian which is at right angles with the cicatrix; in other words, there is a certain degree of ectasia in the region of the cicatrix which probably increases the distance between the ciliary body and the lens margin, and thereby lessens the danger of complete compression of the filtration angle in the future.

An iridectomy may fail in several ways. Profuse hæmorrhage within the eye at the time of the operation, or during the following few days, is perhaps the one over which we have least control. It is chiefly to be feared where the glaucoma is known to have been preceded by intra-ocular hæmorrhage; it may happen in spite of the greatest care where there is advanced degeneration of the blood-vessels; it may be induced by violent coughing, sneezing, or straining of any kind; it may depend on direct injury of the ciliary processes by a deeply placed incision. Its avoidance depends largely on careful regulation of the patient's condition with regard to the action of bowel and kidneys, sleep, and absence of cough; on gentleness in operating, and on rest and tranquillity during the following few days. The worst form of hæmorrhage is the retro-choroidal; it causes extrusion of the lens and vitreous through the incision, and calls for prompt removal of the eye. It is very rare. Failures of any kind through hæmorrhage are quite exceptional. More frequently an iridectomy fails by effecting neither a reopening of the filtration angle nor the formation of a vicarious channel. Thus an incision which lies entirely in the cornea, or nearly so, commonly closes too quickly and firmly, giving an inextensible cicatrix which affords no drainage. Again, a very shallow chamber with high tension involves danger to the lens. As the aqueous escapes the lens is driven forward by pressure from behind, and may be ruptured against the back of the knife through the intervening iris; or without being actually injured, it may be driven forwards so forcibly after the operation as to block the wound and prevent the drainage of the eye; it may even be extruded through the wound during the following twenty-four hours. When, after an apparently perfect operation, we find on the following day that the iris is in contact or nearly so with the cornea, the wound not leaking, and the eye hard, we have the formidable condition known as malignant glaucoma. The displaced lens is blocking the wound, and the best chance of saving the eye lies in making a scleral puncture as described below, and following it at once by steady pressure on the centre of the cornea by means of a curette or other smooth instrument, continued until the anterior chamber is re-established by the entrance of fluid between iris and cornea. For this a general anæsthetic is likely to be required. If this manœuvre fail, or if the lens be injured as well as displaced, extraction of the lens affords a last chance of saving the eye, the posterior capsule being divided at the same time, so as to establish free communication with the vitreous chamber. A shallow chamber after iridectomy need excite no apprehension so long as the tension of the eye remains low.

Sclerotomy.—This operation opens the anterior chamber by an incision more or less resembling the first act of an iridectomy, and leaves the iris intact. Experience has shown that every form of primary glaucoma will yield in some cases to sclerotomy, but it has shown also that sclerotomy is on the whole less trustworthy than the older operation, for the reason that the iris is apt to occlude the wound during the healing process, and that in any case the filtration angle remains more liable to occlusion than after the removal of the iris-segment. The chief use of sclerotomy is as a supplement to iridectomy when the latter fails to permanently reduce the tension. In the absence of the iris-segment it is a simple operation, and is generally preferable to a second iridectomy at the opposite side of the circle. Sclerotomy, like iridectomy, may sometimes be advantageously preceded by a scleral puncture. For glaucoma with deep anterior chamber, as in serous cyclitis and congenital buphthalmos, *paracentesis of the cornea* is usually preferable to iridectomy. In such cases the incision must be of small extent, and repeated if necessary several times, each time at a different part of the cornea. For secondary glaucoma after cataract extraction, sclerotomy combined with division of any membrane which is stretched across the pupil is usually the best remedy.

Scleral Puncture or Posterior Sclerotomy.—The eye is turned inwards so as to expose the outer part of the sclera. The surgeon, taking a Graefe knife in one hand and forceps in the other, seizes the conjunctiva a little below the horizontal meridian, and slides it downwards a little over the sclera. Keeping the back of the knife towards the cornea, and the point directed towards the centre of the globe, he punctures the sclera at a point about six millimetres from the corneal margin and a little below the horizontal meridian. After entering about ten millimetres the knife is rotated on its axis, so as to give a gaping wound through which fluid escapes, and slowly withdrawn. The conjunctiva is then allowed to slide back into its place so that the conjunctival and scleral wounds are not directly continuous. This operation is useful chiefly as a preliminary or adjunct to iridectomy or sclerotomy. Used alone, although it gives immediate relief of tension and corresponding improvement of vision, it rarely if ever effects a permanent cure. A scleral puncture made immediately before a glaucoma iridectomy, especially where the chamber is shallow and the eye very hard, facilitates the making of a good incision, and thereby diminishes the risks and the causes of possible failure which attend an iridectomy under these circumstances. It occasionally, however, reduces the tension of the eye to such an extent as to hinder rather than help the performance of the iridectomy. In such cases, which in the writer's experience are extremely rare, the scleral puncture will suffice for temporary relief, and the iridectomy must be postponed until a more normal tension is restored. In very advanced and doubtful cases, where the possibility of recovering useful sight is uncertain, it is well to make a scleral puncture as a test operation in the first instance, and if the result be encouraging, to make an iridectomy a few days later.

Other Substitutes for Iridectomy.—In cases of annular posterior synechia with bulging iris, it is sometimes impossible to pass the linear knife between the iris and the cornea. It may then be passed through and behind the iris instead of in front of it, and will, in cutting out, re-establish the desired communication between posterior and anterior chambers. Quite a small aperture in the iris may suffice in such a case to permanently relieve the tension.

Incisions and punctures which divide the ciliary region in a more or less meridial direction, and which, if they involve the corneo-scleral junction, will open the aqueous and vitreous chambers simultaneously, were formerly and are still occasionally practised. Such incisions are capable of giving immediate relief and of depleting the ciliary body, but they are not comparable in safety or certainty of result with a well-made iridectomy.

Stretching and rupture of the infra-trochlear nerve, and excision of the superior cervical ganglion of the sympathetic, are methods occasionally employed where other treatment has failed. The evidence in their favour is not very strong.

After-Treatment.—The essential points in the after-treatment are to obtain quietude and sleep, to apply the necessary dressings with little pressure, and to guard against stooping and all sudden or straining movements. So long as the chamber is empty, or the wound obviously leaking, the patient should remain in bed. Finally, the refractive condition of the eye must be tested and corrected with special regard to the astigmatism which the operation commonly sets up, and the patient must be carefully instructed to avoid such conditions or habits of life as might lead to a recurrence of his malady.

LITERATURE.—The literature of glaucoma is voluminous. The following works may be selected for special reference:—VON GRAEFE. *On Iridectomy*, New Syd. Soc. 1859, and papers in early numbers of *Arch. f. Ophth.*—WM. BOWMAN. "Glauc." etc. *Med. Times and Gaz.* 1863.—HERM. SCHMIDT. "Glauc." *Handb. der gesammt. Augenheilk.* 1877.—TH. LEBER. "Interchange of Fluid in Eye," *Arch. f. Ophth.* 1873.—MAX. KNIES and AD. WEBER. Articles in *Arch. f. Ophth.* 1876.—PRIESTLEY SMITH. *Erasmus Wilson Lects.* 1889, and article in Norris and Oliver's *Syst. of Diseases of Eye*, 1898.—SCHWEIGGER. "Glauc." etc. *Arch. f. Augenh.* 1891.—DE WECKER. "On Sclerotomie," *Therapeutique Oculaire*, 1878.—H. SNELLEN. (Historical.) *Ophth. Rev.* 1891.—W. BRAILEY. Papers in *R. Lond. Ophth. Hosp. Reports.*—TREACHER COLLINS. *Erasmus Wilson Lects.*, and papers in *R.L.O.H. Reps.*, and *Trans. of Ophth. Soc.* See also, in latter, a discussion on sclerotomy, etc. 1882.

Glenard's Disease. *See* ENTEROPTOSIS.

Glossitis. *See* TONGUE.

Glosso-Pharyngeal Nerve.

THIS nerve comes out by five or six filaments from the upper part of the medulla, in the groove between the olivary and restiform bodies, and the fibres can be traced to three nuclei, the glosso-pharyngeal nucleus proper, the funiculus solitarius, and some of the fibres, probably motor, pass into the upper part of the nucleus ambiguus, which mainly belongs to the vagus. (Some authorities consider it difficult accurately to differentiate the nuclei of origin of this nerve from those of the vagus, and further hold that this nerve ought to be described as a part of the vagus.) The nerve passes out through the middle division of the jugular foramen, along with the spinal accessory and vagus nerves, but separated from them by a special sheath of fibrous tissue. In the foramen it is external, and also in front of the other nerves, and has on it two ganglia, the jugular and the petrosal, which resemble the spinal ganglia as regards their constituent cells.

Following Quain's description, the nerve appears below the internal carotid and jugular vein, and is directed downwards over the carotid artery, and beneath the styloid process and the muscles connected with it, to the hinder border of the stylo-pharyngeus muscle; then curving forwards, it crosses over the outer surface of this muscle, and passing beneath the hyoglossus ends in branches for the posterior part of the tongue. The jugular ganglion is situated in the jugular foramen, and includes only the lower fibres of the nerve. The petrosal ganglion is situated in a small depression in the petrous part of the temporal bone, and from it the nerve of Jacobson and the branches to the vagus and sympathetic arise.

The nerve supplies *sensory branches* to the back of the tongue, the pharynx, Eustachian tube and middle ear. Taste fibres for the posterior third of the tongue and neighbouring gustatory mucous membrane are carried by this nerve. The fibres probably in most cases leave the glosso-pharyngeal by way of Jacobson's nerve, the small superficial petrosal, and the otic ganglion, to reach the third division of the trigeminus.

The nerve supplies *motor fibres* to the stylo-pharyngeus muscle, and possibly middle constricture of the pharynx, very probably along with the vagus. The nerve also supplies *secretory and vaso-dilator* fibres to the parotid gland through the otic ganglion and the auriculo-temporal nerve, and it is connected with the vagus, the third division of the fifth, the seventh, and the sympathetic.

Etiology and Clinical Features.—The nuclei of the nerve might be injured in the medulla by hæmorrhages, acute softening, sclerosis, or tumours, and at the point of exit of its constituent filaments, by syphilitic disease, aneurysm, or meningitis, and in the peripheral course of the nerve by thrombosis of the jugular vein, or inflammatory swellings, tumours, and so forth compressing the nerve.

The nuclei suffer, but never as an isolated lesion, and in acute and chronic bulbar paralyses other nuclei are always included, and it is hardly conceivable that in a lesion affecting the nerve, whether intra- or extra-cranially, that the vagus at least could escape.

The clinical features can hardly be stated with any degree of certainty, as isolated lesions of this nerve are not obtainable in sufficient numbers to supply data. If the nerve is injured beyond the ganglia then taste should be lost in the posterior third of the tongue. If the ordinary sensory and motor fibres are injured the pharynx will be insensitive, and food is swallowed with difficulty, partly due to this loss of sensation, and partly to the muscles supplied by this nerve being paralysed.

The *prognosis* and *diagnosis* call for no special remark, and the *treatment* must be upon general principles.

Glottis. *See* LARYNX.

Glycosuria.

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See also DIABETES.

THE term glycosuria is applied to that condition in which grape sugar is found *temporarily* in the urine. Thirst and diuresis, and other diabetic symptoms, are usually absent or very slight. Many authors would apply the term diabetes mellitus to all cases in which grape sugar is *permanently* present in the urine (in quantities sufficient to give a reaction to the ordinary clinical tests for sugar). Cases in which a small quantity of sugar is permanently present in the urine, whilst other symptoms are slight or absent, are, however, sometimes described by the term chronic glycosuria; but it appears better to describe such cases as mild forms of diabetes mellitus.

A temporary glycosuria is not usually followed by diabetes mellitus at a later period; but sometimes, after disappearing for a time, the glycosuria recurs, and occasionally becomes permanent, and the condition develops into one of true diabetes mellitus many months or several years after the glycosuria was first detected.

The following are the chief forms of glycosuria:—

1. ALIMENTARY GLYCOSURIA.—In the normal condition the power of sugar destruction of the body is not unlimited, and a very large quantity of saccharine food causes a small amount of sugar to appear in the urine. The various forms of sugar differ in their power of producing alimentary glycosuria. The following are the quantities of several forms of sugar which will produce glycosuria in healthy persons (v. Noorden): milk sugar over 120 grms., cane sugar 150-200 grms., fruit sugar about 200 grms., grape sugar 180 to 250 grms. (the sugar should be given in one dose, on an empty stomach before breakfast). The power of assimilation for starch appears to be unlimited in health. The power of sugar assimilation is sometimes slightly diminished in apparently healthy persons, and is often much reduced in

various pathological conditions (as, for example, Graves' disease, cirrhosis of the liver, many fevers, chronic alcoholism, obesity).

In order to detect a diminution of the power of sugar destruction (alimentary glycosuria) in any individual, Naunyn recommends the following test:—Two hours after a breakfast of coffee and milk (¼ litre), with 80-100 grms. of bread, 100 grms. of grape sugar are given. If distinct glycosuria occurs, then the power of assimilation for sugar is diminished.

Some observers are of opinion that alimentary glycosuria (or *glycosuria e saccharo*) is simply the mildest form of that morbid condition which reaches an advanced state in spontaneous glycosuria (*glycosuria ex amylo*) or diabetes mellitus. Whether this view be correct or not, it has been found occasionally that cases of diminished sugar destruction or alimentary glycosuria (detected by the 100 gramme glucose test as above described) have, in course of time, developed into diabetes mellitus.

Thus v. Noorden found that in 4 out of 15 obese persons, temporary alimentary glycosuria was produced by 100 grammes of glucose. Of these four individuals two became diabetic several years later.

In cases of chronic alcoholism from excessive beer-drinking, Strümpell has drawn attention to the frequency of alimentary glycosuria or diminished power of sugar destruction; and he believes that there is no sharp line between this condition and the true diabetes which is sometimes met with in alcoholic subjects.

In traumatic neurosis or neurasthenia there is often a diminished power of sugar destruction; also Ebstein and Asher have shown that traumatic neurasthenia is sometimes followed by diabetes mellitus.

These facts are sufficient to show that the detection of a diminished power of sugar destruction (or alimentary glycosuria) by means of the 100 gramme sugar test, may be of some practical value with respect to the prophylaxis of diabetes mellitus. Thus, for example, in cases of great obesity, in chronic alcoholism (especially from beer-drinking), in traumatic neurasthenia, and in individuals having a family history of diabetes, if the 100 gramme sugar test shows that there is a diminished power of sugar destruction, it is possible that, by a suitably restricted diet, the development of diabetes mellitus might be prevented.

In such cases sugar and certain articles very rich in starch should be forbidden; also sweet wines and beer should not be taken.

In the urine examinations for life assurance sometimes a trace of sugar is detected, though there is no other evidence of disease. In such cases not infrequently the sugar disappears rapidly, and the urine is normal next day. Here also the 100 gramme sugar test would be of value as a means of showing whether there was any diminution in the power of sugar destruction, and of course, if a positive result were obtained, a modified diet would be indicated.

2. Puerperal Glycosuria (*Lactosuria*).—It has long been known that during the puerperal state a substance occurs temporarily in the urine which reduces Fehling's solution. It has been shown that this substance is lactose in small quantity. It is said to be most abundant on the fourth or fifth day of the puerperium, and it appears to be connected with engorgement of the breasts.

3. Glycosuria produced by various Drugs and Poisons.—(*a*) *Phloridzin diabetes.*—It has been shown by v. Mering that phloridzin, given hypodermically or by the mouth, produces glycosuria with increase in the quantity and specific gravity of the urine, both in man and in animals. These symptoms disappear when the drug is discontinued. According to

some observers there is no excess of sugar in the blood, and the glycosuria is thought to be due to the action of the drug on the kidneys.

(*b*) Other drugs and poisons *sometimes* produce slight glycosuria if given in very large doses; but this result is by no means constantly obtained. Glycosuria has *occasionally* been observed after toxic doses of the following drugs: opium, morphia, chloral hydrate, prussic acid, mineral acids, mercurial salts, arsenic, phosphorus, uranium salts, curare, orthonitro-phenyl-propiolic acid, caffein, theobromine, diuretin, caffein, sulphonic acid, ether and chloroform narcosis, coal gas and carbon monoxide poisoning.

4. Glycosuria associated with various Diseases.—In a number of varied diseased conditions a slight and temporary glycosuria is met with occasionally, whilst in similar conditions it is frequently absent. The following are some of the conditions in which glycosuria has been sometimes noted: injuries to the head, concussion, fracture of the skull, cerebral hæmorrhage, meningitis, brain tumours (very rare except in tumours of the pituitary body, see article on "Diabetes"), Graves' disease, locomotor ataxia (very rare), disseminated sclerosis (very rare), mental diseases (chiefly melancholia), cirrhosis of the liver, obesity, gout; occasionally after acute fevers, such as typhoid, scarlet fever, measles, diphtheria, cholera; occasionally temporary glycosuria occurs during pregnancy. Glycosuria or diabetes is often associated with acromegaly.

(With respect to the relation of gout, syphilis, arterio-sclerosis, and obesity to diabetes and glycosuria, see article on "Diabetes." In this article the forms of glycosuria after removal of the pancreas and after experimental lesions of the nervous system are also described.)

5. Pentosuria.—Recently a number of cases have been recorded in which the urine has reduced Fehling's solution, and the reducing body has been shown to be pentose—a form of sugar containing five atoms of carbon or a multiple thereof in the molecule. No definite symptoms have been associated with the presence of pentose in the urine.

Detection of Glycosuria.—As considerable care is sometimes necessary in distinguishing between small quantities of sugar and other substances occasionally found in the urine, a few words on the clinical tests may be of service. There are numerous tests for grape sugar in the urine, but the three most valuable are Fehling's solution, phenyl-hydrazin, and fermentation. By the employment of these three tests in the following manner and order, as a rule, grape sugar can be easily detected with certainty, even when only a trace is present.

Fehling's solution is the most convenient test. It is important to boil the test solution first: if reddish yellow oxide of copper be thrown down, or if a greenish turbidity be produced, the Fehling's solution has decomposed and a new specimen is necessary. If no change occur the solution is good, and a little urine may now be added, and the mixture boiled. It is important not to add too much urine (never more than the quantity of Fehling's solution employed). Also, the mixture should not be boiled too long. (If these precautions be not taken, a slight turbidity may occur when the urine contains a great excess of uric acid or urates). If on testing with Fehling's solution *no* reaction be obtained, we may safely conclude that the urine is free from sugar in the clinical sense, and *no further testing* is necessary.

If an abundant reddish or yellowish precipitate (copper oxide) occur on boiling the urine with Fehling's solution, the presence of grape sugar may be assumed, providing the precautions mentioned have been taken, and providing other signs of diabetes mellitus be present, such as increased quantity and specific gravity of the urine, diuresis, thirst, etc. But the difficulty arises chiefly when diabetic symptoms are absent, and when the only evidence of disease is the presence of a substance in the urine which reduces Fehling's solution. Often the reduction is slight: it may consist only of a greenish turbidity, occurring when the test-tube cools. Sometimes this slight change is due to sugar, sometimes to other substances which occasionally occur in the urine, and which reduce Fehling's solution, such as glycuronic acid, alkapton, uric acid and urates in great excess, lactose, pentose, etc. Hence some confirmatory test must be employed before a definite opinion can be given. The best two confirmatory tests are phenyl-hydrazin and fermentation.

The following simple method of performing the *phenyl-hydrazin test* is of great service, and if a *negative* result be obtained we can safely state that the substance which caused the reduction of Fehling's solution was not grape sugar. A test-tube of ordinary size is filled for *half an inch* with hydrochlorate of phenyl-hydrazin (in powder); then acetate of soda (in powder or fine crystals) is added for another *half-inch*. The test-tube is half-filled with urine and heated directly over a spirit lamp (*without* the use of a water bath). For two minutes the urine is kept boiling. The tube is then placed in the test stand, and twenty or thirty minutes later, if grape sugar be present, the deposit at the bottom of the test-tube is found, on microscopical examination, to contain needle-shaped crystals of a bright yellow colour. Often the crystals are arranged in sheaves or rosettes. If these crystals cannot be detected, the urine is free from grape sugar in the clinical sense, and no further testing is necessary. An abundant deposit of the crystals in the test-tube is in all probability due to grape sugar. But lactose produces yellow globules with short spines, and glycuronic acid small yellow crystals. Hence when a positive result is obtained with the phenyl-hydrazin test it is always best to employ the fermentation test, which is the most reliable method.

The *fermentation test* may be conveniently carried out in the following manner:—Two ordinary test-tubes of equal size are employed. The same quantity of German yeast (weighed) is placed in the bottom of each (about $\frac{1}{16}$ of the tube is filled with the yeast). One test-tube is filled with normal urine, the other with the suspected urine. The mouth of each test-tube is closed with an india-rubber stopper, perforated with a short piece of glass tubing bent twice at right angles. The two test-tubes are inverted and kept side by side in a warm place, being supported in a glass tumbler. In twenty-four hours, at the top of the test-tube containing the normal urine with yeast, a small bubble of gas will have collected. (This gas is given off by the yeast itself.) At the top of the tube containing the suspected urine with yeast, if grape sugar be present, a greater quantity of gas will have collected. The gas may occupy one-quarter, or one-half, or the whole of the tube, according to the quantity of sugar present. Any excess of gas in the tube containing the suspected urine, beyond the small bubble of gas in the tube containing the normal urine, will indicate grape sugar or other fermentable sugar with certainty,

LITERATURE.—For references on glycosuria, see article "Diabetes mellitus"; also STRAUSS, *Zeitschrift f. klin. Med.* Bd. xxxix. 1900.

Goitre. *See* THYROID GLAND.

Gonorrhœa.—This subject is taken up as follows: the local affections under "Urethra"; the general effects under "Rheumatism" and "Joints"; the results of gonorrhœal infection in the female are the subject of a separate article on "Gonorrhœal Infection."

Gonorrhœal Infection.—Although the special inflammations resulting from an attack of gonorrhœa have been considered under their appropriate headings, it will be convenient to summarise the effects produced by gonorrhœal infection in the female.

In the acute form the vulva, vulvo-vaginal glands, and urethra are involved, accompanied by swelling of the inguinal glands and even abscess formation. The vagina is stated by some authorities to be rarely affected, but this depends entirely on the character of the epithelial lining. In children the vagina is affected. In adults where connection has occurred frequently, the vaginal walls offer more resistance to the action of the poison. A secondary vaginitis is not infrequently produced through the discharge from the cervix uteri, causing desquamation of the superficial layers of the vaginal epithelium. Œdema of the labia is a most important sign, accompanied in many cases by superficial ulceration. The urethral orifice is inflamed, and on pressure on the urethra with the finger introduced into the vagina a purulent discharge exudes.

The distinction between a gonorrhœal attack and one due to other causes is chiefly the degree of inflammation, and previous to the discovery of the gonococcus the severity alone was relied upon to settle the diagnosis. Now, however, the discharge should be examined microscopically to discover the presence or absence of the special micro-organism. In acute cases there is further inflammation and erosion of the cervical mucosa, from which a copious purulent discharge flows. The systematic examination of the discharges in cases of gonorrhœa has demonstrated the important influence of the cervical discharge, not only in acute, but also in chronic gonorrhœa. In the latter group of cases the cervical discharge remains infective for a lengthened period.

Where suitable treatment has been employed the disease may be cured before it has spread farther, and the more serious complications averted. If however, douching, the incautious use of the sound or other instrument, has caused an upward extension of the poison, the body of the uterus, the ovaries and Fallopian tubes, the pelvic peritoneum, and cellular tissue become in turn involved in the disease. Owing to the changes produced in the ovaries and Fallopian tubes sterility is a frequent consequence. Well-marked examples of what has been termed acute ascending gonorrhœa are found in puerperal cases, where infection has occurred late in pregnancy, and where the changes in the genital tract produced as a result of the delivery of the child favour the upward extension of the gonorrhœal poison. During pregnancy, owing to increased secretion of mucus and epithelial desquamation, the vagina is more liable to be affected.

The severity of the attack in acute cases prompts the sufferers to seek medical relief. In the subacute and chronic cases, where the symptoms are slight, the disease is allowed to progress untreated, unless some further complication arises. There is further the difficulty in persuading the class of patients who suffer from gonorrhœa to continue treatment until they are completely cured. Such patients with a gonorrhœal discharge from the cervix uteri continue to be sources of infection to those with whom they come in contact.

When a case of endo-cervicitis comes for treatment, it is well to examine the discharge for gonococci, for, as already mentioned, the passage of the sound or other instrument favours the upward extension of the gonorrhœal poison. It should be added that the periodic congestion resulting from the menstrual functions tends to keep up or aggravate the cervical catarrh. There is considerable difficulty in estimating the proportion of cases of salpingo-oöphoritis which are due to gonorrhœa. Statistics have been published by several German writers with a view to determining the exact proportion. The statement that $\frac{1}{5}$th of the cases is due to gonorrhœal infection is probably correct. Septic infection, the result of a confinement, a miscarriage, or "persistent local treatment!" to the cervix uteri, and tuberculous infection, are answerable for the remainder.

The clinical features of the cases of ascending gonorrhœa, with implication of the Fallopian tubes and ovaries, will be best understood by describing a typical example:—

A healthy young woman has married a man who had contracted gonorrhœa six months previously, and who believed he was cured. Three or four days after marriage his wife complains of pain and soreness on passing water, accompanied by vaginal discharge. The intensity of the initial manifestations may vary considerably, but the history of definite local signs and symptoms can usually be obtained. The vaginal discharge persists long after the dysuria has disappeared. Menstruation, which before marriage had been regular and almost painless, soon becomes irregular and preceded by considerable pain. The pain commences gradually in the intermenstrual periods, and is aggravated by standing or exertion. It is felt not only in the hypogastric and iliac regions, but also in the sacral region, and extending down the thighs. The pain may or may not be relieved by the menstrual flow.

The patient soon begins to lose flesh, and is unable to do any work, and, as the severity of the symptoms increases, may be entirely confined to bed. She becomes a chronic invalid, dragging on a weary existence, never free from aches and pains. During the progress of the illness the pain is liable to acute exacerbations, accompanied by elevation of temperature, thirst, nausea, and actual vomiting. Such attacks are frequently termed by patients "inflammation of the bowels," but are, in reality, due to pelvic peritonitis, spreading from the Fallopian tube, or in the severe cases actual leakage of pus through the abdominal ostium before it has become occluded. Sterility develops as a consequence of the tubal disease.

On *abdominal examination* either no swelling may be detected, or a swelling in the iliac regions, tympanitic on percussion due to adherent intestine, which may in bad cases form a roof to the pelvis, thus rendering an abdominal operation very difficult. Where large collections of pus have formed in the tubes these may be palpated per abdomen.

Vaginal examination reveals a mass situated on one or both sides of the uterus posteriorly. If unilateral, the uterus is pushed towards the sound side, and will probably be movable with the mass. The vaginal roof on the affected side is depressed. If bilateral, the masses, when of large size, may interfere with the mobility of the uterus, that organ being fixed in the centre of a bilateral swelling. The vaginal roof on both sides is depressed. The masses in the pelvis are either the tubes and ovaries matted together by adhesions, or definite purulent collections in the tubes or ovaries, or in both.

Where definite purulent collections exist, the pus must be evacuated. The method of doing this need not be discussed here. The treatment of the cases where no purulent collection exists has led to much controversy. The results of operation are disappointing; pain frequently persists; whereas the expectant treatment by rest and suitable remedies has given fairly good results, and should always be tried before an operation is recommended. Complete recovery may follow when gonorrhœal salpingitis has been properly treated. It is erroneous to suppose, as some writers do, that all cases require operation.

A word may be said concerning the sterility which results from gonorrhœal infection. There may be absolute sterility, but more frequently the sterility is relative (the "one-child sterility" of Sänger). The phenomenal sterility of prostitutes is probably due in part to the presence of adhesions fixing the Fallopian tubes and ovaries, and, further, to atrophic changes occurring in these organs as a result of preceding disease. Many prostitutes have had one child, and are afterwards sterile. The birth of the child may have been the cause of their adopting this mode of life, or it may have favoured the upward spread of the gonorrhœal poison.

The diagnosis of gonorrhœal cases, where the history is indefinite or unreliable, is always difficult apart from bacteriological tests. If, however, a patient who shortly after marriage develops symptoms as detailed in the illustrative example, and who has not had any local treatment or vaginal examination, nor any miscarriage or confinement, and in whom tubercle can be excluded, the cause is probably gonorrhœa.

"LATENT GONORRHŒA."—Many writers have persisted in stating that an attack of gonorrhœa cannot be completely cured, and although apparently cured, tends to recur after alcoholic excess or sexual indulgence.

Where treatment has been ineffectual or neglected, the discharge may persist, but with suitable remedies the disease can be thoroughly eradicated. Ever and anon cases occur which are capable of producing gonorrhœal infection for two or more years. I know of no case definitely authenticated in which the pus contained gonococci after four years.

In the majority of the very chronic cases reported the proof of the presence of gonococci is incomplete.

Noeggerath,[1] whose statements are much exaggerated, insisted on the importance of what he termed "latent gonorrhœa." He believed that a man is never completely cured of an attack of gonorrhœa, and that that man is certain to infect his wife.

It is further supposed that in cases of so-called "latent gonorrhœa" indulgence in alcohol or marital intercourse brings to life certain gonococci which are lying in a dormant state.

If an attack of gonorrhœa in the male remains uncured, the affection is increased by indulgence in alcohol or by sexual intercourse. This, however, is a case of chronic or neglected gonorrhœa, not one of so-called latent gonorrhœa.

After a severe attack, even when the disease has been cured, a discharge, purulent or mucoid, may result after alcoholic or sexual indulgence. In such cases, however, no gonococci can be demonstrated in the pus.

Where a patient has had several attacks of gonorrhœa the urethra becomes structurally altered, and although these attacks confer a certain immunity as regards the action of the gonococcus, the urethral mucosa becomes unduly sensitive to the action of other pus organisms. Any increased congestion or irritation may give rise to a purulent urethritis. No gonococci are found in the pus.

The great advantage which has accrued from Noeggerath's work is that the medical profession, and through it the public, have been educated to the importance of having an attack of gonorrhœa completely cured. Further, the suffering and misery which attends gonorrhœal infection in the female has been demonstrated by other workers, and patients are now treated in the early stages, and the diseased cured before important internal organs are involved.

The suggestion of the term "latent gonorrhœa" has led to a deal of what may be termed speculative pathology, which has been ridiculed by those opposed to the influence of micro-organisms in disease. It would be well to discontinue this term, as such cases are better named uncured, neglected, or chronic gonorrhœa.

The Character of the Infection which causes the Complications and Sequelæ of Gonorrhœa.—This is a difficult question to decide, and in the present state of our knowledge we are unable to arrive at definite conclusions.

The possibilities may be briefly stated as follows:—

(1) Pure gonorrhœal infection, the result of the invasion of gonococci alone.

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[1] *Die latente Gonorrhoea in weiblichen Geschlecht.* Bonn, 1872.

(2) Mixed infection, the result of the invasion of pus organisms, which have developed in the mucous surface originally attacked by the gonococci.

(3) Secondary infection, where the gonococci produce the complication, followed later by the entrance of pus organisms, both existing together until gonococci die or are overgrown by the pus cocci.

(4) Toxic infection—the complications may be the result of the absorption of toxic products produced by the gonococci or the pus organisms.

Under one or more of these headings the complications and sequelæ of gonorrhœa may be explained.

It is probable that a certain amount of toxic infection is present in every case.

From the writer's own observations he is led to conclude that the effect of the action of the gonococcus on the particular soil attacked is to convert that soil into a suitable medium for the growth and development of pus organisms.

If the invasion of pus organisms be great we may have various complications developed quite apart from the action of the gonococcus.

In the consideration of this subject we may be led astray if, after constantly finding the same collection of organisms in certain lesions, we fix on one species as specific, and neglect the others as contaminations.

The majority of the complications in the female are due to a mixed infection or to a secondary infection. The cases of rapid upward spreading of the disease, attended by the formation of pyosalpinx, are in all probability examples of secondary infection. The more chronic cases are rather examples of mixed infection.

The more efficient drainage for the female discharges renders the occurrence of marked toxic phenomena less frequent. Where, however, the outflow is obstructed, as in certain cases of pyosalpinx and ovarian abscess, then toxic phenomena do occur. The presence of such phenomena, however, depends entirely on the virulence of the purulent contents, as many cases of pyosalpinx and ovarian abscess are unattended by much systemic disturbance, not even elevation of temperature.

Although it is stated that articular complications are relatively rare in women, it will be found that in the majority of cases a history of joint pains can be elicited. The occurrence of effusion into one or more joints is not uncommon, and ankylosis may develop as a consequence. More frequently, however, the joint affections are not severe, consisting in pain on movement accompanied by little or no effusion. The possibility of gonorrhœal infection as a cause of synovitis in women should always be remembered. In the lower extremity the knee and ankle joints are most commonly affected. In the upper extremity the wrist-joint is selected.

Treatment.—This is discussed under the Complications of Gonorrhœa, and only a short summary need be given here.

In acute cases rest in bed should be advised, and in addition the avoidance of injections, which undoubtedly are very harmful.

The writer does not recommend douching even in chronic cases, as in both the treatment can be more effectually and thoroughly done by swabbing out the vagina through a speculum with strong silver nitrate or carbolic acid solution. A similar application may be made to the endo-cervix, and not carried into the body of the uterus. In acute cases an anæsthetic may be required owing to the pain and difficulty in introducing a speculum. If, however, this treatment by swabbing out the vagina be properly carried out the onset of the disease may be checked.

Too frequently the patients have used douches, or have been advised to use them, with the result that the upward extension of the disease has been increased.

Now that gonorrhœa has been proved to be due to a microbe whose effects are at first purely local, involving mucous membranes, it follows that the rational treatment consists in an early destruction of this microbe.

For this purpose a germicide is required which has the power of soaking into the mucous surface. Nitrate of silver and strong carbolic acid possess this power, and are the best applications.

If the immediate treatment of gonorrhœal infection was more efficiently employed, less would be heard of those serious forms of ascending gonorrhœal inflammation.

In pregnant women a thorough swabbing out of the vagina will prevent the upward spreading of the disease after the confinement, and will save the eyes of the child from infection at birth.

In addition to rest in bed and avoidance of injections the bowels should be kept freely open by saline aperients. Alcohol should be forbidden. For further details the reader is referred to the special articles "Vaginitis," "Vulvitis," etc.

Medico-legal Aspects of Gonorrhœal Infection.—The discovery of the gonococcus was at once hailed as of importance in medico-legal work, *e.g.* in cases of rape where gonococci were discovered in the vulvo-vaginal discharge of the victims when the accused suffered from gonorrhœa.

This is certainly an important corroboration; still bacteriological science is not yet sufficiently exact to warrant our making very definite statements. There are still many important points requiring elucidation before we can reach that degree of accuracy required for expert evidence in a court of law.

A great controversy has existed for some time, and still exists, concerning the nature of the vulval discharges in infants and young children. Indeed, their occurrence has been used as a foundation for false charges of rape against innocent individuals. In such cases a careful examination should be made of the vulva, noting the presence or absence of excoriations, and in addition the discharge should be microscopically examined to determine the presence or absence of (*a*) spermatozoa, (*b*) gonococci. The chief difficulty surrounds the identity of the gonococci, for various Continental authorities have stated that these cases of vulvo-vaginitis in infants and young children are all due to gonorrhœa,—some being caused by direct infection, while others are due to infection from dirty towels, linen, etc.

In this relation it must be remembered that there is a popular superstition that sexual intercourse with a virgin will cure an attack of gonorrhœal urethritis.

A series of cases of vulvo-vaginitis in children were carefully examined by the writer of this article, with the result that although a few are gonorrhœal the great majority are not.

The reason why so many conflicting statements have appeared in papers written on this subject is that, as the writer believes, mistakes have been made over the identity of the organism present.

In the vulvo-vaginitis of infants and young children the discharge frequently contains an organism resembling closely in form and colour reactions the gonococcus, but differing in this important point, that it can be cultivated on gelatine, whereas the gonococcus does not grow on this medium.

It is this organism which has been mistaken for the gonococcus, and has led to the erroneous statements which have been made on the etiology of vulval discharges in children.

The identity of any micro-organism is not complete until the most reliable evidence of all, viz. the cultivation test, has been furnished. If then the purulent discharge be examined microscopically, and the organisms cultivated, a more correct view of the causation of this disease will be obtained. It will be found that a large number of the contributions on this question may be disregarded, as in the investigation of the subject the cultivation test was not employed.

In countries where prostitutes are systematically examined, bacteriological tests should be applied to the discharges before declaring that a woman is free from infection. The statistics furnished from various large Continental cities show how frequently gonorrhœa is found among women of this class. The bacteriological tests (microscopic and cultural) require to be oft repeated, many specimens of the discharge being examined, and at different periods, before a definite opinion is given.

LITERATURE.—Noegerrath. *Die latente Gonorrhoe in weiblichen Geschlecht.* Bonn, 1872.—Bumm. *Der Mikro-organismus der gonorrhöischen Schleimhaut-Erkrankungen*, 1885, Wiesbaden.—M'Cann. "The Ætiology of Gonorrhœa," *Transactions of the Obstetrical Society of London*, vol. xxxviii.—Bumm. "Die gonorrhöischen Erkrankungen der weiblichen Harn und Geschlechtsorgane," *Veits Handbuch der Gynäkologie*, vol. i. p. 427 *et seq.* 1897. (A very complete list of the literature is given with this paper.)

Gout.

Fr. *Goutte* ; Ger. *Gicht.*

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See also Rheumatism, Rheumatoid Arthritis.

Introduction.—It has been truly said that if a sprightly writer wished to make fun of the medical profession, nothing would give him a better opportunity for the exercise of his sarcasm than the extreme variety and the very opposite character of the regime which this, that, and the other medical authority have laid down for the dietetic treatment of the gouty state. Some recommend a diet mainly vegetarian in character, others find salvation in meat and hot water, and, apart from these two extremes, when we have regard to the numerous articles of food and drink in daily use, we find very few that are not as stringently forbidden by some writers as they are highly commended by others. The reason for this is readily found in our ignorance of the exact nature of the disease. After ages of inquiry we know gout only by the order and character of its phenomena, and have yet to learn its intimate nature or the special cause which produces it. We know so very few definitely established facts of importance in the etiology of the disease that a doubting practitioner may well be pardoned when he asks if we know any. But, in proportion as the facts are few, the hypotheses are numerous, and the too ready acceptance of conclusions, lacking confirmation in generally accepted facts, has been responsible in great measure for retarding our knowledge of the disease.

As we cannot yet give the physiological history of gout, we should adopt a pathology founded solely on facts unalloyed by hypotheses, and conformable to all that we know of the physiology of animal life. In the course of our inquiry as to the appropriate treatment it cannot fail to be remarked how much this treatment is dependent on the same general principles which guide us in other diseases, and how little consideration it admits for the special nature or character of gout.

There are two points that should ever be before those who work or write on this subject. All clinical experience teaches us that we are dealing with a constitutional disease whose natural history includes a liability to rapid and apparently inexplicable variations, often fortunately of the nature of amelioration or complete disappearance of symptoms. If these natural variations occur coincidently with the use of some supposed therapeutic measure, medicinal or otherwise, there is a risk of the observer wrongfully ascribing the benefit to the therapeutic agent employed, when it is in reality due to the self-righting power of nature. He becomes what Sir W. Roberts aptly termed the victim of misinterpreted sequences. Another fruitful source of error is the laboratory. Only too frequently have measures which may have appeared on theoretical grounds to be applicable to the treatment of the disease succumbed before the searching light of practical experience. The fallacies in experimental laboratory work are so numerous that great care must be exercised in drawing any conclusions from this line of inquiry, and should the conclusions arrived at not be in harmony with the teachings of clinical experience they may well be abandoned.

A few words about the term *uric acid diathesis*. Under this term are generally included two conditions—gout and lithæmia—which have only one important point in common. In both the excretion of free uric acid in the urine plays an important part. The frequent association of these two conditions, either in the same individual or in different members of the same family, has not unnaturally led to the belief that there is an exceedingly close relationship between them, even to the extent of regarding uric acid calculi as gouty deposits in the urinary tract. While we must admit the probable existence of a very close relationship between the two conditions, they yet present sufficient points of difference in their appearances and results as to justify us considering them separately. The etiology and treatment of uric acid calculi will be considered later (see "Uric Acid"); for the present we are concerned with gout proper. The subject is admittedly a complex and difficult one, and while its adequate presentation involves frequent entry into unknown and purely speculative regions, I shall seek to indicate them clearly, so that we may avoid arriving at any conclusions not based on definitely ascertained and accepted facts.

MORBID ANATOMY.—The characteristic anatomical changes in gout are dependent on the deposit of urate of soda in various parts of the body, notably in and around joint structures, and on secondary changes arising from this deposit. Much discussion has from time to time taken place as to whether degenerative changes occurred in the tissues prior to the deposit of the urate compound. While some observers maintain that such degeneration is a primary factor (Ebstein and his school), others hold that urate of soda can be deposited in unchanged living tissues (Riehl, Lukhatschiff, and His). The subject is a complicated one, and requires further elucidation. While the uratic deposits are seen most characteristically in joint structures they are by no means confined to them. Nearly all the structures of the body which contain a large amount of connective tissue have been found affected. In the central nervous system, crystals of urate of soda have been found in the dura and pia mater, in the neurilemma of nerve-sheaths, in patches of cerebral softening, and in the cerebro-spinal fluid. A similar condition has been noted in the cardio-vascular system, the aortic and mitral valves and aorta occasionally showing a deposit. Among other sites may be mentioned the eyelids, sclerotic, auricle, tendons and tendon sheaths, vocal cords, bronchi, bursæ, bone marrow, palmar, and other fascia, and subcutaneous tissue generally. Indeed it is probable that a careful examination of the various organs and tissues in pronounced cases of gout would reveal the presence of a deposit in many other places not previously described. These points sufficiently indicate that uratic precipitation is very variable in its incidence, a fact of considerable import in connection with the phenomena of irregular gout. Before considering the changes in articular structures a few words may be said about the deposition of urate of soda.

The presence of urate of soda may be manifest as a subcutaneous infiltration of the connective tissue and tendon sheaths (*e.g.* in Dupuytren's contraction), or it may be present in the form of localised subcutaneous swellings—tophi. In yet other cases it may be a component part of an enlarged, thickened, and inflamed bursa, in which case the swelling may reach a considerable size. *Tophi* vary in size from a very minute deposit, the size of a pin's head, up to or exceeding that of a small orange. Among the more common situations are the ears, fingers, foot, ankle, and eyelids, but they may be seen in other situations. The general appearances vary according to the site and the presence or absence of ulceration. Their consistency and chemical composition are also variable. The term chalky deposits usually applied, while giving a fair general impression of their appearance, is not strictly accurate, as there is no carbonate of lime in their composition.

Comparatively few detailed observations have been made on their chemical composition. The results obtained by Langin, Wurzer, and Lehman are given in the following table :—

	Langin.	Wurzer.	Lehman.
Sodium urate	25·92	29·70	52·12
Calcium urate	15·75	29·30	1·25
Sodium chloride	16·70	18·00	9·80
Potassium chloride	...	2·20	...
Calcium phosphate	...	...	4·32
Animal matter	16·70	19·50	28·49
Water Undetermined residues	19·60	10·30	3·88

Lehman's specimen was supposed to include a fragment of bony substance. Calcium oxalate has also been found in large amount. The salts of uric acid, which are the important ingredients, can be readily recognised by the following reactions :—Take a fragment of the tophus and dissolve by the addition of a few drops of nitric acid. Evaporate slowly almost to dryness in a porcelain capsule, when, if uric acid be present, a yellowish deposit appears, and on addition of one or two drops of ammonia a rich purple colour is exhibited—purpurate of ammonia. This constitutes the well-known murexide reaction.

When present in the subcutaneous tissue in a diffused form the uratic deposit may not be readily observable to the naked eye, but its presence may be suspected from the associated thickening and contraction of the tissues, and by coincident ulceration. A correct diagnosis may only be determined by a microscopic or chemical examination, the former revealing characteristic needle-shaped crystals of urate of soda, the latter being readily determined by the murexide test.

The Joints.—When a gouty joint is examined in the earlier stages of the disease the articular cartilages show scattered or isolated points, streaks or patches of a "chalk-like" material—urate of soda. A closer examination may reveal the fact that this deposit is not really on the surface of the cartilage, but is situated interstitially in its substance, the superficial layer of epithelium being intact. Later this superficial layer is involved, and the articular surface becomes roughened, irregular, and eroded. As the primary deposits take place in the areas where circulation and nutrition are at their lowest level, we accordingly find that it is the central portion of the articular cartilage that shows the earliest manifestations. As the disease progresses the ligamentous structures become the seat of interstitial deposit, and in inveterate cases they may become extensively destroyed. Before this has taken place, however, the synovial membranes and their fringe-like processes are involved, the surface of the synovial membrane appearing as if dusted over with powdered plaster of Paris. The rest of the synovial membrane is usually congested, thickened, and thrown into irregular folds. The synovial fluid may be in excessive amount, and may be turbid from the presence of specks of urate. An acid reaction has been observed in one instance. In very rare instances the contents of the joints have been found hæmorrhagic or purulent. The ends of the bones frequently become enlarged, and true bony ankylosis occasionally occurs. Changes also take place outside the joints, the connective tissues, aponeurosis, and tendon sheaths becoming the seat of a varying amount of uratic infiltration. The bursæ in the neighbourhood of a joint may be extensively involved independently of any joint affection. Secondary changes are induced in the joint structures by its occurrence. Inflammatory changes occur in the cartilage leading to proliferation and necrosis, the proliferative changes being most manifest along the borders of the cartilage where the deposits are smaller and the tissues more highly vascularised. As a result we have outgrowths at the margin of the articulation, best seen in the larger joints. Some degree of osteitis also occurs with degenerative changes in the bone and marrow, and a varying degree of uratic infiltration. Wynne, quoted by Duckworth, holds that the hypertrophic outgrowths (lipping) present in gout must be regarded as true exostosis, and not ecchondroses as in rheumatoid arthritis.

The microscopic appearances of the cartilages are fully described by Duckworth, who is of opinion that there is no microscopic appearance of cartilage characteristic of gout.

Before leaving the consideration of the joint changes attention may be drawn to an important contribution by Moore on the subject of uratic deposits in joints. Moore (*St. Bartholomew's Hospital Reports,* 1888) made an extended series of observations into the condition of the joints, small and large, in over seventy post-mortems. In some of the cases recorded no gouty history had been obtained during life. Two of his conclusions have a very special bearing on the points under consideration:—

(*a*) It is common to find urate of soda in the joints of those persons whose aortic valves show chronic degenerative changes with calcification, and who therefore belong to the class of patients likely to have had angina pectoris.

(*b*) Urate of soda is present in the joints of a large proportion of those persons over forty years of age who die of cerebral hæmorrhage.

The importance of these observations is not perhaps sufficiently recognised in this country, where they serve to justify a diagnosis of gout in not a few cases where joint changes are entirely latent. But they are of still greater importance from the point of view of the opinions held by various foreign writers on the too frequent diagnosis of gout by British physicians. While admitting that a diagnosis of gout is frequently arrived at on insufficient grounds, these observations prove that it is not advisable always to withhold a diagnosis of gout (and substitute rheumatism) except in cases where there has been a previous acute attack, or where there is distinct clinical evidence of uratic deposits.

VISCERAL CHANGES IN GOUT.—The only visceral disease which is intimately related to gout is contracted granular kidney, and the relationship is a complicated one. In some cases the renal disease precedes the articular manifestations; in others it seems to appear as a sequel, and in a few the conditions appear and develop simultaneously. Moore found that chronic interstitial nephritis was not invariably accompanied by deposits in the articular cartilages. Ord and Greenfield found that in a series of cases of gouty affection of the great toe joint, in two-thirds of them there was a definite coexistence of contracted granular kidney, and in the remaining third there was an affection of the kidney closely allied to it. The question is a difficult one, the difficulty being partly due to the fact that whereas a diagnosis of chronic renal disease is frequently arrived at without much difficulty, in other cases this is not so, and it may be impossible to affirm that the kidneys are not diseased. Gouty kidneys may show a few marked deposits of urate of soda both in the cortex and medulla, the deposit being situated in the intertubular connective tissue, and only occasionally in the tubules.

Typically gouty deposits are also occasionally met with in the heart (once), arteries, and veins, and in other situations; but as these are rare, and the associated cardio-vascular diseases are very common, no great stress can be laid on their occurrence. In the course of the disease numerous other morbid changes affecting different structures and organs are met with, but as they are referred to under chronic gout they do not call for detailed reference.

GOUT IN LOWER ANIMALS.—It is obvious that if positive results could be obtained in the production of gout in lower animals, very valuable information might be gained from them regarding the etiology and treatment of the disease. So far, however, observations on these lines have been too limited to be of much practical value.

The investigations of Žalesky and others, which were originally undertaken about the year 1850 with the view of elucidating the part played by the kidney in the formation of uric acid, gave results which agreed in demonstrating the presence of deposits of uric acid salts in various organs and tissues.

In 1882 Ebstein carried out an elaborate series of experiments, the results of which led him to formulate the theory associated with his name, viz. that necrotic changes in the tissues are the primary cause of gout, the necrosis being due to the presence of dissolved urates in the fluids of the body. One set of experiments consisted in the ligature of the ureters of cocks; a second series comprised the subcutaneous injections of small doses of neutral potassium chromate. By the first means he prevented the elimination of the urates, and so led to their being dammed back and retained in the tissues; the potassium chromate was regarded by him as exercising a damaging influence on the renal epithelium, in virtue of which its excretory activity was impaired, with the result that a similar although less acute retention of urates occurred. The fowls died within twenty-four hours after the ligature of the ureters, but lived for a few weeks after chromate administration. Post-mortem examinations in both instances revealed the presence of uratic deposits in the articulations, tendon sheaths, liver, muscular tissues, and serous membranes, these changes being much more pronounced in the chromate experiments. Ebstein, however, considered that the differences were those of degree only, and corresponded to the different lengths of time the animals survived. It has been urged against these experiments that, as uratic deposits were prominent in the liver, muscular tissues, and serous membranes, localities which are not similarly affected in human gout, the two processes cannot be regarded as wholly comparable. While the possible validity of this objection must be recognised, we must also admit the possibility that the conditions found by Ebstein are really analogous to human gout, the difference being only one of degree.

In 1888 Mendelson published an interesting paper on guanin gout in the hog and its relations to the sodium urate gout of man. This subject had been primarily referred to by Virchow and Roloff, but the observations of these observers were not elaborated. Mendelson found numerous small discrete, chalky-looking masses of guanin in the periosteum of bones, and on the surface of the junction between the epiphyses and the shafts. Similar deposits were abundant in the ligaments and peri-articular tissues generally, and also scattered indiscriminately in the cartilage covering the ends of the bones and semilunar cartilages. The muscles showed like deposits, especially in the intermuscular septa. In no instance were any signs of inflammatory reaction about the site of deposit discovered, which circumstance, considered along with the absence of structural change in the tissues immediately around the areas of crystallisation, led Mendelson to conclude that there was a very gradual accumulation of the deposit, allowing the tissues to adapt themselves to its presence. This observation led him to the belief, in opposition to Ebstein's view, that the deposit was primary and the necrosis a secondary development. This guanin gout is an exceedingly rare condition. It may be identified as follows:—Evaporated upon platinum foil with a drop or two of strong nitric acid, a slimy, yellowish red residue remains, which, if touched when cold with a drop of sodium or ammonium hydroxide solution, becomes of a deep reddish brown colour, changing to dark purple on heating (Hoppe-Seyler). This test is quite distinct from the murexide reaction of uric acid. I am informed by large importers that American hams sometimes show the presence of minute specks suggestive of the description given by Mendelson, but so far I have not been able to discover any specimens.

The latest recorded experiments by Kossa and Kionka have been more dietetic in nature. Kossa in 1899 fed birds on cane sugar and dextrose, and succeeded in inducing so-called avian gout. This consisted in the appearance of crystals of sodium urate in the looped tubules of the kidney, followed sooner or later by renal inflammation. There also developed in the tissues a condition similar to that described by Ebstein in his chromate experiments. He also found a marked increase in the nitrogenous metabolic products, and a point of some interest therapeutically was determined by him, viz. that the animals so affected died all the sooner if they were treated with piperazin.

The administration of oxalic acid and its salts, corrosive sublimate, acetone, and other substances, also induced these appearances of avian gout, although in a milder form. With regard to dogs and rabbits, it was observed that the subcutaneous injection of sugar did not produce a similar result.

In 1898 Minkowski, by feeding dogs with adenin, a substance closely allied to uric acid, induced a deposit of urate of soda in the kidneys. His observation is a suggestive one, and a confirmation and extension of this line of inquiry is desirable. At present, however, the difficulties in the way of procuring adenin are almost insuperable.

The most recent and probably the most interesting experiments yet recorded are those of Kionka (1900). After a preliminary reference to the occurrence of avian gout in fowls, ostriches, and birds of prey, he goes on to describe his own experiments. These consisted in feeding full-grown fowls on a diet of minced horse flesh (freed of fat and gristle) and water. He found that after a period of three to five months a disease developed which gradually assumed the characters of real gout. Several types of disease were observed. Some fowls exhibited a more rapid form of the disease, characterised by weakness of the lower limbs, loss of appetite, unsteady gait, and swelling of the joints, these symptoms being aggravated at intervals in a manner suggesting acute attacks. Death occurred eventually from exhaustion. On post-mortem examination the joints were swollen, œdematous, and showed deposits of urates. These deposits were found to be more pronounced in the more chronic cases, in which the attacks had not been so manifest, and where the most marked features were the development of tophi in the joints and webs of the feet. In other cases the features of visceral gout were manifested, uratic deposits being present in the serous membranes of the intestine, and uric acid infarcts in the kidney. In all the cases the usual appearances post-mortem were those of gouty kidney.

From some points of view it is unfortunate that a more detailed account of the histological characters of the organs and tissues affected, and a fuller record of the end products of metabolism, are not given in this interesting paper. This line of inquiry is one of great interest, and as it is important that the observations be repeated and extended, I have arranged to do so, and am at present engaged with it. At the present time, after nearly two months' treatment, the fowls under observation are not obviously gouty, although there are indications that some of them are not in perfect health. The results of the investigation will be described in the article "Uric Acid."

Gout is usually described, although in rather vague terms, as one of the rarer diseases of a poultry yard. I have taken some pains to obtain information from the leading poultry experts in this country, but cannot get unequivocal evidence in reply. One expert writes: "A swollen condition of the feet, attended with heat, to which fowls are liable, has been wrongly described as gout. Such a condition is simply due to congestion of the blood-vessels consequent on a plethoric state of the system, and birds in confined runs, when exercise is limited, or practically prevented, are very susceptible to stagnant circulation in the limbs and feet, which produces these supposed gouty symptoms." On the other hand another writes:—

"This (*i.e.* gout) is rather liable to be mistaken for leg weakness, but may be distinguished by the legs and feet feeling hot, with evident swelling, and a more or less inflamed appearance. It is chiefly found in Asiatic breeds. The bird should be removed to a dry warm place, and given a dose of jalap or calomel to open the bowels, after which a half-grain pill of extract of colchicum should be administered twice a day. The legs and joints may be well rubbed with sweet oil daily with benefit." Other references bear out this latter statement. I make no apology for entering so fully into this matter, as it is one of practical importance, especially as the reader may have opportunities of testing the accuracy of these statements, and so add to our knowledge of the subject.

ACUTE GOUT.—The manner of onset, course, and subsequent history of acute gout varies within such wide limits that no description, however detailed, can be made applicable to each individual case. Peculiarity of constitution, whether hereditary or acquired, continually varies the aspect of the disease, the natural course of which is doubtless also undergoing invisible yet none the less important modification under the influence of improved hygiene, greater muscular activity, and the slightly improved habits of eating and drinking of the present day compared with those obtaining in the past.

With regard to a description of the acute attack I cannot do better than give Sydenham's graphic description, based as it is on direct personal knowledge of the disease as well as on extensive clinical experience otherwise. This description is minute and accurate even at the present day, it is historically of great interest, and the little admixture of theory shown in the reference to digestion and dispersion of the peccant matter does not deduct from the value of his clear and interesting record:—

"Suddenly and with scarcely any premonitory feelings the disease breaks out. Its only forerunner is indigestion and crudity of the stomach, which troubles the patient for some weeks previous to the acute attack. His body also feels swollen, heavy and windy, symptoms which increase from day to day until the fit breaks out. A few days before this torpor comes on, and a feeling of flatus along the legs and thighs. Besides this there is a spasmodic affection, whilst the day before the fit the appetite is unnaturally hearty. The victim goes to bed in good health and sleeps. About two o'clock in the morning he is awakened by severe pain, generally in the great toe; more rarely in the heel, ankle, or instep. This pain is like that of a dislocation of the bones of those parts, and is accompanied by a sensation as of chilly water poured over the veins of the suffering joint. Then follow chills and shivers and a little fever. The pain, which was at first moderate, becomes gradually more intense, and while it increases the chills and shivers die out. Every hour that passes finds it greater, until at length at night time it reaches its worst intensity, and insinuates itself with most exquisite cruelty among the numerous small bones of the tarsus and metatarsus, in the ligaments of which it is lurking. Now it is a violent stretching and tearing of the ligaments, now it is a gnawing pain, and now a pressure and tightening. So exquisite and lively meanwhile is the feeling of the part affected that it cannot bear the weight of the bed-clothes nor the jar of a person walking in the room. Hence the day is passed in torture, and a restless rolling, first to one side then to the other, of the suffering limb, with perpetual change in posture; the tossing of the body being about as incessant as the pain of the tortured joint, and being at its worst as the fit is coming on. Hence the vain efforts, by change of posture both in the body and the limb affected, to obtain an abatement of the pain. This goes on towards the second or third hour of the morning (a whole day and night after the first outbreak of the fit), such time being necessary for the moderate digestion and dispersion of the peccant matter. The patient thus has a sudden respite which he falsely attributes to the last change of position. A gentle perspiration is succeeded by sleep.

"He wakes freer from pain, and finds the part recently affected swollen. Up to this time the only visible swelling had been that of the veins of the affected joint. Next day (perhaps for the next two or three days), if the generation of the gouty matter has been abundant, the part affected is painful, getting worse towards evening and better towards morning. A few days later the other foot swells and suffers the same pain. The pain in the latter regulates the state of the one first attacked, for the more acutely it is tortured, the more perfect is the abatement of suffering and the return of strength in the other. Nevertheless there is a repetition in the second case of all the misery of the first, both as regards intensity and duration. Sometimes during the first days of the disease the peccant matter is so exuberant that one foot is insufficient for its discharge. It then attacks both and that with equal violence. Generally, however, it takes the feet in succession."

With regard to *premonitions* there is a great diversity in the nature and frequency of their occurrence, and in a few cases none may have been observed. Those most frequently encountered have reference to the digestive system, the usual run of symptoms in a robust adult being as follows:—

The patient does not feel well, but cannot clearly define his feelings. He feels "gouty" and thinks you should understand the feeling. On closer inquiry we find that there has been some slight irregularity in the intestinal action, a little undue peristaltic movement associated with slight discomfort not amounting to pain, slight constipation, a moderate degree of flatulence, with fitful sleep the preceding night. The tongue is slightly furred posteriorly with a faintly yellowish white fur, the breath is a little foul, the appetite unimpaired or capricious, the conjunctivæ a little muddy, and the pulse tension slightly raised. If he has had many previous attacks, there are other subjective sensations in the arms or legs. His facial expression reflects the coming trouble, and, as the mind is preoccupied with the analysis of these various disturbances, some depression of spirits or irritability of temper is a not unnatural sequel. In other cases various nervous disorders are the most prominent indications — shooting pains, cramp, neuralgia, headache, cardiac irregularity, and irritable temper.

The *constitutional disturbance* is as a rule out of proportion to the amount of febrile movement. The temperature does not usually rise above 102°, but varies with the severity of the local inflammation, and to a less extent with the number of joints involved. The fever remains from three to five days, but may remit and recur. The acute attack may occur at any period of the day, and while the usual course of the disease is for the great toe joint to be the part first affected, in not a few cases the primary attack occurs in the knees, ankles, tarsus, or hands. This fact may be explained in some cases by the previous occurrence of an injury to the part affected, in virtue of which its vitality has been permanently weakened, and the part rendered more prone to the incidence of the existing poisons. In other cases, however, there is nothing discernible either in the previous history or occupation of the patient to explain this departure from the usual course of the disease. When the swelling increases the pain lessens; later the parts pit on pressure, and the cuticle cracks and desquamates. At the height of the attack the pulse tension is usually high, the tongue heavily coated, the appetite gone, hiccough, eructations, sometimes vomiting, and also constipation present. In very severe cases, where the affection has ceased to be a purely articular one, I have observed that pain in the interior of the bones was a distinct feature. Only very exceptionally does suppuration attend the gouty inflammation. The state of the blood and urine is referred to later (p. 219).

While acute gout is commonly a disease of adult life, not appearing as a rule under the age of thirty-five, there are numerous exceptions to this. In cases where acute manifestations of the disease develop at an earlier date, from the late teens onwards, the paroxysm is less declared; not infrequently all that is manifest is a slight attack of monarticular pain, with redness, swelling, and distended veins, lasting for a varying number of hours. Such cases may develop and recur without ever culminating in the classical picture of an acute paroxysm. In severe cases of confirmed and recurring gouty manifestations of an acute nature, metastasis is of very frequent occurrence.

The *subsequent history* of an attack varies within wide limits, and is mainly dependent on the hereditary quality of tissue in each individual. The more speedily the constitutional disturbance is concluded the less is the risk incurred of the gouty diathesis becoming confirmed, and the greater the security against the associated ailments entailed by protracted or oft recurrent gout. If appropriate treatment be not assiduously carried out the disease tends to recur, and acquiring force by repetition eventually inflicts its wonted penalties. Attacks may recur every year, or even much more frequently, and indeed there is no limit to the irregularities in the frequency or severity of their occurrence. As a favourable illustration Sir W. Roberts described a case where the first attack, a typical one, occurred at the age of twenty-nine, and the next one in the patient's eighty-ninth year.

The *subsequent local changes* similarly vary. Sir Dyce Duckworth has described a case in which careful examination of the joint previously affected revealed no evidence of gouty deposit. On the other hand, we know from Moore's observations that gouty deposits frequently exist in joints that have not been recognised clinically to have been affected with gout.

In very severe and chronic cases the weakness in a limb, recovering from an acute attack, is very considerable, and may only be partially recovered from after several months. Such cases are usually accompanied by muscular wasting, and possibly also by nerve degeneration, and are usually seen in patients whose natural vigour, originally weak, has been much reduced by the ravages of the disease.

CHRONIC AND IRREGULAR GOUT

It is probably inadvisable to attempt to differentiate these two conditions too closely. To illustrate. One man has a first attack of genuine articular gout when æt. 20, and in five years or so he is almost a cripple from the disease. Another man may have the first acute manifestations when æt. 28 or 30, and have ten or twelve typical acute attacks before the age of 40, and yet at that age there may be no external evidence of articular or other abnormality. A third man æt. 40 may have suffered from well-marked symptoms of irregular gout for ten or fifteen years, but has never experienced an acute attack, and, like the preceding case, may show no distinct external evidence of the disease. The difference between these three clinical types is one of degree and not of kind. If we selected at random a dozen fairly illustrative cases of the last-mentioned group, and experimented with them as with the lower animals, with the object of inducing typical acute attacks, we would probably be successful in most if not in all the cases. The degree of success would be proportionate to our skill in attaining, without *other unsought constitutional disturbance*, the environment in the digestive tract and in general cell metabolism appropriate to the development of the disease, and the failures would be explained either by faulty methods of experiment, or, occasionally by special idiosyncrasies of the patient. The experiment would doubtless be of further interest as showing different modifications of the acute phases of the disease, corresponding more or less to the great variety in the irregular manifestations originally present.

Just look for a moment at a few of the more important factors which modify the appearances of chronic and irregular gout. These are—

(i.) The force of heredity, and the purity of the gouty strain. The last is a factor of very far-reaching importance.

(ii.) The state of the digestive tract, *e.g.* is it tolerably good, as it often is in cases of pure gout, or is it naturally more or less seriously weakened by hereditary weakness (gouty or otherwise)? The latter group furnishes a familiar class of cases, usually thin, more or less dyspeptic-looking individuals with symptoms mainly gastro-intestinal or nervous. The weak digestion seems to act in these cases as a natural safeguard against the more acute articular manifestations.

(iii.) The state of the nervous system is an all-important factor in the disease, and especially in its irregular development. A recent clever novelist, in referring to the early demise of one of her feminine characters, recorded the death as due to the combined effects of ennui and luxury. Neither alone, the authoress states, would have been sufficient to kill her, but she could not stand against the allied forces. A fairly accurate lay-picture of not a few cases of irregular gout.

The severity of the symptoms is by no means proportionate to the extent of the gouty deposits. This is well illustrated in case 4, p. 246, where the deposits were very pronounced, and yet gave trouble mainly through their bulk. In other cases the deposits are scanty and the symptoms are severe. It has been suggested that these varieties depend on the fact that the amount of disturbance induced depends more on the site than on the extent of a deposit.

Keeping these different factors in mind, it is obviously impossible to present anything like a complete picture of the chronic and irregular phases of the disease, and it must suffice to give merely a short outline of chronic gout, including the deforming and tophaceous variety with the associated cachexia, and thereafter an equally brief outline of the phenomena of irregular gout as seen in the various systems of the body.

Chronic Deforming Gout.—Here the articular changes are very pronounced, and are usually most marked in the joints of the fingers, ankles, knees and great toe, but they are by no means confined to these structures. There is also found uratic infiltration with degeneration in various fasciæ, bursæ, and tendons in the neighbourhood of the affected joints, and indeed in not a few cases these sites are the primary ones. In a few instances the greatest deposit may be in one or other of the important bursæ in the region of a joint, and such cases may be less amenable to treatment than those in which there is a more diffuse articular change. The deformities induced are those due to the swelling of the joints, deflection of the fingers, hands, and toes, ankylosis and other local changes, and they closely simulate the appearances in rheumatoid arthritis. (See " X-Rays.") *Nodi digitorum*, the term applied to the knotty or knobby state of the terminal phalangeal joints by Heberden (Heberden's nodes), are frequently of a gouty nature.

Chronic Tophaceous Gout is characterised by deposits in the subcutaneous tissues, notably of the ears, in the neighbourhood of joints, and in various bursæ. These tophi may give way and exude a characteristic creamy fluid. They may be present in large size in the neighbourhood of a joint, which otherwise appears free from the disease and whose mobility is unimpaired.

Gouty cachexia is the sequel of chronic gout in certain predisposed subjects, but the causes of that predisposition are not quite clear. It is seldom met with in the robust, plethoric subjects who present the classical pictures of the disease, and is more usually seen in cases where the disease develops early in subjects strongly predisposed to it. The symptoms of the cachexia are numerous and varied, but in the main are those of cachexia from any other cause. The anæmia and associated degenerative changes in the muscular and nervous tissues in turn react unfavourably on the gouty tendency. The functions of all the muscular, glandular, and nervous organs are profoundly deteriorated, and the results in any given case vary according to the extent of the degenerative changes in one or other or all of these organs. The prognosis is bad when the cachexia is doubtful.

IRREGULAR GOUT—GOUTINESS

The symptoms of irregular gout are manifold and varied, and are not infrequently most developed in persons who have never been the subject of the acute phases of the disease, and in those who have no pronounced evidence of chronic gout. Are we justified in the diagnosis in these cases? Certainly.

While we must avoid ascribing all diseases occurring in subjects with a hereditary history to a gouty origin, a careful study of the heredity, constitution, and clinical features in many cases will amply warrant the diagnosis. The views entertained by not a few writers that a diagnosis of gout is not possible in the absence of tophi or a history of previous acute attack are not to my mind tenable. But a careful discrimination must be exercised in this direction.

To what are the symptoms of irregular gout due? Can they all be explained by the view that they are dependent on the local deposit of minute crystals in the affected parts, *e.g.* gastric mucous membrane, cerebral membranes? Or are they dependent in whole or in part on the selective action of a soluble toxic substance in the blood on a naturally weak spot? A final answer cannot yet be made. The symptoms vary with the age and constitution of each subject, but in every case we may take it that the symptoms are more liable to develop in that system or tissue which happens to have, from natural or acquired defects, the weakest nutritional activity. Here again we must have regard to the influence of other hereditary strains in modifying the direction and severity of the gouty symptoms.

THE NERVOUS SYSTEM

We are only beginning to realise how often gout acts on many organs and tissues, and how frequently inflammation is thus induced (Gowers and Taylor, 1899). This opinion well illustrates the importance of the association between gout and diseases of the nervous system, and at the same time it indicates the limited extent of our precise knowledge of the subject. The effects of gout are seen both on the higher and lower neurones. *Neuritis* is by no means infrequent, and is probably most encountered in the third and fourth decades of life, but it is probable that many cases of supposed neuralgia in young subjects are really minor forms of neuritis. Sir Willoughby Wade was of opinion that a neuritis of the nerves at the affected part was a feature of the acute attack, but I have never found the distinct linear tenderness described by him. Some time ago, while experimenting with electrical currents on the hand of a gouty friend, I was much interested to find that the cutaneous sensibility in a finger whose joints were the seat of articular changes was much diminished. The point is worthy of further investigation.

Myelitis is rare. Gowers considers that possibly some cases of otherwise undetermined origin in young adults owe their origin to this cause. In *neuralgia* we find the most common, as it is also one of the most troublesome, nerve derangements. Its relationship to gout, and its association with other traits of the disease, are frequently perfectly apparent and unmistakable, and would probably be more so if sufficiently detailed inquiries were instituted. The fifth nerve, posterior tibial, sciatic, and occipital are the ones most frequently involved, and their derangements are liable to appear and disappear suddenly, or take the place of other manifestations of the disease. Cases like the following are instructive and by no means rare:—A young man æt. 32, of plethoric build and hair gray almost to whiteness, came complaining of "acidity." His father had been a martyr to gout and in his later years was affected with attacks of angina pectoris. When 25 years old this patient had a very severe attack of neuralgia which fully prostrated him for two or three weeks, and incapacitated him from work for as many months, and at the end of this time his hair was "almost white." Lesser attacks have occurred subsequently, and, curiously enough, the prodromal symptoms were the same as those experienced by his father —a feeling as if the right thigh was being sponged over with cold water. From the great severity and long duration of the first attack it may probably be regarded as a regular acute gout in an unusual site. There is indeed no end to the irregularity and anomalous nature of the nervous disturbances encountered. In young adults *tender feet* may be an early symptom, and is usually accompanied by the hot sensations in the feet described by Duckworth. The discomfort arising from this condition in young women may be so great that they will never wear boots or outdoor shoes a minute longer than they can possibly help. In one such case I have observed a localised point of exquisite tenderness over the middle of one calf, and this patient averred that the feelings of *cramp* to which she was subject seemed to have their origin at this point. This case was specially interesting, as the patient was by no means of a neurotic temperament.

The *local phenomena of Raynaud's disease* are also frequently seen in the subjects of inherited gout, the pallor and coldness of the affected extremity persisting from a few minutes to half an hour or more. These patients are usually very susceptible to the influence of cold water, and cannot tolerate sea-bathing. In adult life *vaso-motor instability* may be a prominent feature, especially in plethoric subjects.

Headache, migraine, insomnia, and *vertigo* are also frequently encountered as the leading subjective indications of gouty metabolism, and they may be accompanied by feelings of physical and mental lassitude, or melancholia. All these cases may be associated with some obvious aberrations in urinary excretions. At and after the climacteric we sometimes find that patients who have been treated for years for recurring acute and subacute gout of a typical character, recover completely from the symptoms, but develop more acute *mental derangements,* necessitating appropriate and permanent asylum treatment. Are these cases to be regarded as illustrations of metastasis, or as illustrating a reversion to another type of inherited weakness under the influence of the diminishing metabolic activity of later life?

The liability of many gouty subjects to *cerebral hæmorrhage* need only be referred to, and it may be well to mention that in old-standing cases of hemiplegia the deposit of urate of soda takes place more readily in the joints and tissues of the paralysed side.

CIRCULATORY SYSTEM

In early life the subjects of inherited gouty proclivities frequently exhibit various vascular disturbances, *e.g.* chilblains, and the local phenomena of Raynaud's disease. In later life the leading manifestations are the following:—

(i.) General vaso-motor instability.

(ii.) Incidental and more or less frequent aberrations in cardiac action, arising from the influence of the toxic agents on the nervous mechanism of the heart, *e.g.* arrhythmia, tachycardia, bradycardia, angina. The tendency to these disturbances is, of course, increased if there be myocardial degeneration.

(iii.) The symptoms and physical signs arising from the action of a hypertrophied heart, with or without dilatation, and contraction of the peripheral vessels, in cases with renal cirrhosis and widespread arterial disease.

(iv.) Evidence of early or late cardiac muscle failure, due to chronic myocarditis, with disease of the coronary vessels.

These various symptoms are frequently induced or aggravated by reflex injurious influences from the gastro-intestinal tract, and in many of these cases the dietetic treatment is the all-important one. This is specially true of the senile heart. Pericarditis is by no means an uncommon manner of termination in cases where the gouty form of Bright's disease is developed. In addition, various arterial and venous disorders are met with. Arterio-capillary fibrosis, and atheroma and their sequelæ, are the arterial derangements; phlebitis and thrombosis are the leading venous disorders. Varicose veins in the legs and hæmorrhoids are by no means infrequent. While the diagnosis of a gouty origin in many of these cases is often largely a matter of inference from the *tout ensemble* of the case, the fact of the occasional relationship of these symptoms to gout should not be lost sight of.

ALIMENTARY SYSTEM

The gastro-intestinal tract is in the closest sympathy with the cutaneous system, and there is every reason to believe that the various nervous and vascular disturbances in the latter are represented by analogous changes in the internal continuation of the epithelial covering. There is no doubt that some of the most troublesome cases of irregular gout are those whose symptoms are referred to the digestive tract, and nowhere more than in this system do we find good illustrations of the doctrine of metastasis. The nature of the symptoms varies considerably in different cases. In one case they are sudden in onset, sharp and fugitive in nature, and give place to definite articular or other manifestations. For example, a sudden *gastralgia* with associated derangement of the digestive functions develops, persists, it may be, for a few days, and suddenly disappears, coincidently with the development of an eczema, an arthritic attack, or an urticaria, and *vice versa.* Acute attacks of abdominal pains (*enteralgia*), with a varying amount of constitutional disturbance, are also seen in undoubtedly gouty subjects, and the clinical appearances and course under treatment may leave no room for doubt as to the accuracy of the diagnosis.

Apart from these more purely nervous manifestations, catarrhal states of the mucous membrane are prone to occur. *Pharyngitis,* acute or chronic, is the most common, but gastro-enteritis is also met with. The very acute congestion and swelling of the pillars of the fauces, uvula, and tonsils, often with distension of the superficial veins, form a quite characteristic picture. Parotitis is a rare incident in the gouty, and when present seriously interferes with mastication and deglutition. It usually develops suddenly, persists for a few hours, and disappears.

In the more asthenic type of the disease the *dyspeptic symptoms* are more those of chronic enfeeblement of the digestive powers, the more important signs being epigastric pain or discomfort, attended by waterbrash and slight flatulence ("acidity"), some depression in spirits, and other evidences of deranged metabolism. This class of case is specially liable to show some obvious urinary abnormality, *e.g.* lithæmia or phosphaturia, which conditions may alternate.

In other cases we find slight attacks of gastric catarrh, usually termed biliousness, characterised by loss of appetite, furred tongue, foul breath, and constipation.

While we must admit that all these symptoms are commonly seen independently of gout, this should in no way interfere with our recognition of their gouty origin in many cases. A few authentic cases have been recorded which revealed unmistakable post-mortem evidences of uratic deposit in the alimentary tract, and such are probably present, though in very small amount, to a greater extent than has been described.

And further, we must bear in mind that the uratic deposit is a late sequel and an inconstant feature of the disease. Its presence is conclusive proof of its existence, but its absence in no way negatives it. We must remember the incessant nature and also the rapidity of the chemical changes occurring in the digestive tract, the liver, and tissues generally. If the views put forward under etiology are correct (p. 223), the relationship between various foods, liquids, etc., and gouty symptoms becomes a little more clearly defined and understood.

A careful study of the general and hereditary history and clinical features of each case will usually suffice to determine its exact nature, and the more thoroughly various minor disorders are studied, the more evidence will they frequently afford of their origin from a common stock.

To what extent are the so-called functional diseases of the liver associated with gout? Alike from the important action of the liver on the carbohydrates of the food, and in the formation of urea (*vide* "Liver, Physiology of"), we have ample evidence in support of the view that in gout, and perhaps even more particularly in irregular gout, liver derangement plays an important part. But the exact nature of the derangements in function induced by temporary vascular and other alterations is quite unknown. It is complicated and probably also inconstant, and with our present knowledge, we must be satisfied with a few general reflections gained from empirical treatment (*vide* "Liver, Functional Disorders of"). This will be further referred to under "Uric Acid."

The last point that calls for reference is constipation. Gouty subjects are much more prone to the injurious effects of constipation of even a slight degree than non-gouty individuals.

RENAL SYSTEM

The hereditary influences of gout frequently show their earliest indication in connection with the urinary system. Gibbons, in an interesting paper,[1] has drawn attention to the frequent occurrence of renal colic in infants of gouty stock. This is further emphasised by the following cases lately recorded to the writer by a medical friend, himself a martyr to gout. His personal history is as follows: Acute gout, twice, in ball of right great toe, gouty dyspepsia, dry eczema on skin, grooved and fissured nails, and one joint of right hand deformed and ankylosed. Three of his children have exhibited symptoms. The youngest, when an infant, was severely troubled with disordered micturition, and bladder irritation, with passage of much free uric acid; another was troubled when a boy with hæmaturia due to the passage of large quantities of uric acid; and a third has had an attack of acute gout in the heel, and in his youth was affected with erythema nodosum. When considering the subject of uric acid excretion in infancy I may draw attention to the statement usually made in most works on pediatrics as to children passing relatively more uric acid than adults. So far I have not been able to trace this to any definite series of observations, and lately I made an investigation on the excretion in a child of 20 months, and the results (as yet unpublished) indicate that the proportion of uric acid to urea in the infant is not essentially different from that in the adult. The point is one that merits further investigation.

Transient albuminuria is met with in gout, especially during the acute attacks, and the amount and frequency of its occurrence may be regarded as evidence of the degree of renal weakness. Similarly transient glycosuria occurs, and may be regarded as similar evidence of hepatic (and possibly also muscular) insufficiency. If pronounced, it may merge into true diabetes.

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[1] *Med.-Chir. Transactions*, vol. lxxix. 1896.

Oxaluria is a less frequent occurrence. Phosphaturia is by no means rare, and an apparent excess of phosphates occasionally alternates with the passage of large amounts of free uric acid. But it should be carefully noted that the occurrence of oxaluria or phosphaturia often depends entirely on the state of acidity of the urine. If the acidity of the urine is low (sodium phosphate present in small amount) there is a greater tendency to the deposit of the oxalic acid normally present in the urine, and there is also a preponderance of the neutral, not readily soluble, and basic, easily soluble, earthy phosphates over the acid salts. We are not yet in a position to state authoritatively that phosphaturia is to be regarded as an indication of abnormal disturbance in the phosphorus metabolism in the organism.

EAR

The external ears, and the parts around, are a favourite site for the development of gouty eczema, and the auricle is prone to the deposit of tophi. Beyond these two facts there are few definitely determined as conclusive of the relationship between gout and ear diseases, but there are numerous isolated references bearing on the point. Gout is said to exercise considerable influence in the development of exostosis in the external auditory canal (Pritchard). Hang has described a case where the ear was affected by prodromal pains, with hyperæmia and swelling of the auricle for several days, followed on the third or fourth day by an attack of articular gout. Mirk has recorded chalky deposits in the tympanic membrane, and he is of opinion that some cases with subjective noises are due to gouty deposits in the labyrinth. Baum also testifies to the effect of gout as a cause of ear diseases, especially in producing earache at night and tinnitus aurium without deafness. In the absence of more fully detailed clinical and post-mortem records of the condition of the middle and internal ear, we cannot speak authoritatively on the influence of gout on the structures of the middle and internal ear.

EYE

There is no mistaking the important part played by gout in eye affections, although only in a few cases have definite deposits of urate of soda been recorded in the conjunctiva (Garrod) or elsewhere. Conjunctivitis, episcleritis, sclerotitis, iritis, and irido-cyclitis are the most common manifestations, and the worst feature in these conditions is their great liability to recur. In the gouty form of iritis, hæmorrhage into the anterior chamber is not infrequent (Maitland Ramsay). Gouty subjects are more prone than others to suffer from glaucoma, and Jonathan Hutchinson has recorded cases of hæmorrhagic retinitis and optic neuritis which he regarded as of gouty origin. Apart from these obvious disorders, pain in the eye may be a prominent symptom. This takes the form of a sudden acute pain, sometimes described as linear in nature; in other cases there may be a dull ache with slight tenderness or hot and itchy feelings in the eyeball. In these latter cases the ocular tension should be carefully investigated. The eyelids are also prone to be the site of a fugitive œdema.

CUTANEOUS SYSTEM

French writers in particular lay great stress on the special liability of gouty individuals to skin affections, the arthritides, as they call them, but in our own literature we find much less belief in the importance of this relationship. Its great importance is undoubted. It would be strange indeed if this extensive vascular connective tissue covering did not react strongly in many ways to the influence of gout. Every experienced family practitioner knows that this is so. He may have seen at one time a severe attack of abdominal pain disappear with the onset of an eczema, or the development of a troublesome and persistent eczema in a limb already the seat of acute or subacute articular gout; or a gouty family of three, one of whom suffered from headache with lithæmia, a second was troubled with supra-orbital neuralgia and eczema, and a third with erythema and nail disturbances. These and the like convince him, from his own experience, that there is a profound relationship between skin affections and gout. Again, he has known of a lichen developing, and persisting in spite of all kinds of appropriate local treatment for years, possibly undergoing slight modification coincidently with the development of some articular or other lesion, which for the first time suggested that the two conditions had a common origin. At another time he has been strongly inclined to diagnose scarlet fever in a boy (in the Christmas holidays), but, from his knowledge of the delicate, sensitive skin, both in the patient and other members of the family, also from his knowledge of the heredity, and consideration of the dietetic habits incident to the time of year, taken in conjunction with the itching present, he finally commits himself to a diagnosis of dermatitis, and in a few days there developed the feeling characteristic of exfoliative dermatitis.

These are all actual occurrences, and many other suggestive parallels could be cited. Similarly with *some cases* of pruritus, urticaria, psoriasis, and herpes, and indeed his whole clinical experience, judiciously expressed, clearly establishes for him the importance of the relationship. One reason for the smaller amount of attention given to the subject in this country may arise from the fact that primary cutaneous disorders are more frequently found in subjects who are the victims of less declared gout, and too much stress is laid on the absence of so-called definite gouty symptoms. A careful study of many skin diseases from the point of view of their relationship to a diathetic tendency is a present desideratum, and an essential basis for the inquiry is a full recognition of the significance of the various minor and irregular manifestations of the disease, along with a zealous and critical investigation of the hereditary history of each case.

The view advanced by Roberts that, owing to the tolerance of the skin for uratic deposits, the relationship between gout and eczema is less important than frequently believed, must be received with caution. The uratic deposits are an incidental and often very unimportant symptom of the disease, and it is even conceivable that their presence in considerable amount may be a safeguard against any cutaneous disturbances in the immediate neighbourhood, owing to the development of morbid structural changes in the vascular and nervous cutaneous supply, and the secretory glands at the part. Gouty skin eruptions are seen in their most typical forms in cases of chronic or irregular gout, with few external evidences of the disease, and they are probably very closely related to the circulation in the vessels of the skin of faulty end products of nitrogenous metabolism (probably not uric acid), and to a lesser extent to the attempts of the cutaneous glands to excrete these abnormal substances. The importance of external influences must not, of course, be overlooked. They constitute an important allied force.

Respiratory System.—As definite deposits of urate of soda have been found in the vocal cords, arytenoid cartilages, and in the crico-arytenoid ligaments and joints, and crystals of uric acid have been reported in the sputum, we are justified in concluding that many minor symptoms of respiratory disorders occurring in subjects predisposed to gout are in reality of gouty origin. With regard to *catarrhal states of the larynx* our knowledge is well summed up in the following. Whether or not there be a specialised gouty form of chronic laryngitis there can be no doubt that in certain persons of plethoric habit, limited diet, abstention from alcohol, and the administration of Carlsbad salts, hasten recovery, as pointed out by von Ziemssen (M'Bride).

The part played by the bronchial mucous membrane is even more pronounced. A *simple irritability of the bronchial mucous membrane*, evidenced by a loud spasmodic cough, is occasionally a well-marked feature in the female children of gouty parents. Occasionally an *acute bronchitis* develops coincidently with the abrupt cessation of articular pains, more especially in inveterate cases in whom emphysema and a proportionate degree of heart weakness are present. In other cases the bronchitis may give place to some other manifestation. The relationship of gout and *asthma* is an interesting and intricate one. Asthma occurs by no means infrequently in the offspring of gouty parents, and in such cases its occurrence must be regarded as a typical gouty manifestation (we must, of course, have regard to the possible strong admixture of a neurotic strain in the individual). While some cases are by no means readily amenable to treatment, others furnish the most brilliant illustrations of the influence of a physiological diet and a regulated life on the faulty metabolism. In some cases, with a long-standing asthmatic history, true articular gout may develop in late life; in others a typical gouty attack may alternate with a genuine asthmatic seizure; perhaps in the majority no other well-declared phenomena take place. In this latter instance it would almost appear as if one outlet for the action of the products of the deranged metabolism sufficed, and even gave some protection against others. Lastly, we must refer to the occasional occurrence of *hæmoptysis* in subjects with a gouty tendency, either declared or latent, this being associated with marked emphysema and vascular degeneration, a condition first described by Sir A. Clark. In all such cases care must be taken to eliminate a chronic tuberculous lesion as the cause of the hæmorrhage. While gout and active tuberculous disease are not often associated, we are not, to my mind, justified in thinking that there is any antagonism between them. Everything depends on the predominance of one or other strain. It is interesting to note that M. Baumis considered that a gouty father and a tuberculous mother will beget an asthmatic child. The chart at page 224 shows that the oldest son of a gouty mother by a tuberculous father was a confirmed asthmatic.

Reproductive System.—The female pelvis is rich in connective tissues, its vascular supply is considerable and subject to periodic variations, and its nervous system is more than usually important and complex. Surely we have here a happy hunting-ground for the manifestations of a disease whose end products are deposited in connective tissues, and in whose life-history nervous and vascular disorders occupy a prominent place. Yet this does not seem to be the case. A reference to the indices of not a few authoritative gynæcological works either reveals no mention whatever of the disease or the barest possible reference to it. One cannot help wondering whether this position is arrived at after a careful study of the possible influences of diathetic conditions on the pelvic viscera, or whether the possible importance of such influence is entirely overlooked. The admitted difficulties in connection with its study hardly warrant the latter position. "If the disorders of the uterus and its appendages were studied more particularly with reference to diathetic conditions I am of opinion that some new chapters in their pathology might be written" (Duckworth). This opinion is well worthy of the careful consideration of those who have the special clinical experience necessary to advance our knowledge of this part of the subject.

MUSCULAR AND CONNECTIVE TISSUES.—The palmar fasciæ and less frequently the plantar fasciæ are occasionally the seat of gouty disorders (see Dupuytren's contraction under "Fascia" in vol. iii.), and Jonathan Hutchinson has drawn attention to a juvenile form of Dupuytren's, in which the induration and contraction are limited to one or more digits, and does not involve the palm. The same author has also drawn attention to the induration in the fibrous structure in the dorsum of the penis as being allied to Dupuytren's contraction, both conditions having a relationship to inherited gout. It may be well to refer here to the occurrence of nocturnal priapism in elderly males affected with irregular gout.

Probably some cases of localised myalgic pains, usually regarded as muscular rheumatism, are in reality of gouty origin. Lumbago is so to be regarded in a proportion of cases, and the aches and pains, of a dull and aching or of a sharp and fugitive character, experienced from time to time in the neighbourhood of joints or other tissues which have been the site of injury at an earlier period, are in all probability of a similar nature. The important part played by the structures in bursæ has already been referred to.

ETIOLOGY

Previous to the year 1793 the views entertained as to the etiology of the disease are only of historical interest, and a full and interesting account of them will be found in the various works referred to in the literature. In that year Murray Forbes suggested that gout developed through an excess of uric acid depositing itself in the tissues. A few years later (1797) Wollaston demonstrated the presence of uric acid in gouty deposits, and from that time until the present, the existence of a definite and important relationship between gout and uric acid has never been seriously questioned. But as to the exact nature of this relationship we at the present time know little more than the opinion expressed by Murray Forbes more than a hundred years ago.

In 1850 Garrod made his classical contributions to the study of the disease. His most important observation was the discovery of uric acid in the blood, but many of his other observations—clinical, pathological, and therapeutic—yet rank as authoritative even at the present day, and all writers and workers on the subject are under a deep obligation to his standard work. For an adequate appreciation of his work the reader is referred to his treatise on the subject. Here I can only indicate a few points of importance with regard to his views on the etiology of the disease, and more especially those dealing with the acute paroxysm. Recent methods of investigation have lately shown that some of his conclusions require revision and modification. From his researches he formulated the following conclusions regarding the acute attack:—

i. The alkalinity of the blood is lowered.
ii. The amount of uric acid in the blood is greater than in the interval.
iii. The excretion of uric acid falls, this being associated with uric acid retention in the tissues, depending on a diminution of the excretory capacity of the kidneys.

These three conclusions formed the basis of his theory of the causation of the phenomenon of the acute attack.

In 1898 the views as to the diminution of the alkalinity of the blood and to the increased amount of uric acid present in the blood during the acute attack were seriously called in question by Magnus Levy. This observer made an extended series of observations in seventeen cases of gout, using the most modern and reliable methods, and in no case did he find evidence of a diminution in the alkalinity of the blood or of an increase in the amount of uric acid as compared with the interval between the attacks. Levy's observations on these points appear to be conclusive. With regard to the third point the writer has recorded a series of observations, the results showing an actual increase of uric acid excretion during the attack. Similar observations have been made by Pfeiffer, Levy, Badt, His, and others. Taken collectively, these investigations seem to prove that the three aforementioned conclusions are erroneous, and they therefore indicate that Garrod's views as to the etiology of the acute attack must be abandoned. If this is so, we must start afresh in search of the cause of the acute paroxysm.

In his later writings Garrod adopted the view that the kidneys are the seat of formation of uric acid in health, an opinion which must obviously very materially influence the views put forward as to the etiology of gout. In this view he has of late years been ably supported by Luff, who has formulated the following conclusions from his original observations on the subject:—

i. Uric acid is not normally present in the blood of man and other mammals, nor in the blood of birds.
ii. Uric acid is normally produced only in the kidneys.
iii. The presence of uric acid in the blood in gout is due to its deficient excretion by the kidneys, and to the subsequent absorption of the non-excreted portions into the blood from these organs.

This view as to the physiology of uric acid is in opposition to those entertained by most physiologists, and in addition the writer has published a series of observations which tend to negative these conclusions. His investigation comprised the examination of very large quantities of the blood of birds, and also the liver, kidneys, and spleen, and the results showed the presence of an appreciable amount of uric acid in the blood and also in these various viscera.

In 1882 Ebstein, as the result of an elaborate series of experiments already briefly referred to, came to the conclusion that the essential element in the gouty process was the development of necrotic areas in the tissues, in which crystalline deposits of urate of soda subsequently took place. He regarded the tissue necrosis as the primary condition, and the uratic deposits as a secondary development. He accepted Garrod's view that the uric acid was the causal agent of the necrotic process, his opinion being that the tissues were led to undergo necrotic changes by contact with uric acid *in a soluble form*. The increase in the uric acid was regarded by him as arising in the majority of cases of primary articular gout through excessive formation of uric acid, and he considered that it was only in the rare cases of primary gouty kidney that retention of uric acid occurs. He considered that the excessive formation of uric acid took place in abnormal situations in the body, especially in the muscles and medulla of bones, and he further held that acute or chronic conditions, or even permanent organic changes, may occur if the excessive uric acid in the lymph passages of the skin, bones, or muscles become congested, as it may do in various parts of the body at once. If the congestion in the lymph passages leads to cessation of lymph flow, then there occurred the sudden acute typical attack of gout.

Many objections have been raised to Ebstein's views. Thus the retention of uric acid produced in birds by ligature of the ureters is very much greater than the uric acid retention in man in acute gout. Further, the acid reaction of the necrosing tissues recorded by Ebstein as essential for the deposition of the uric acid salts is not borne out by v. Noorden, who repeatedly found an alkaline reaction of softened tophi. We must also have regard to the opinions of various observers already indicated, to the effect that deposit of urate crystals takes place in *living* tissues, a view diametrically opposed to Ebstein. And lastly, Luff has suggested that, as in Ebstein's experiments, uratic deposits occurred in the muscles, liver, and serous membranes, situations very rarely encountered in the human subject, the two conditions are hardly to be considered analogous.

V. Noorden has advanced the view that the uric acid formation and deposition is a secondary process produced by the presence of a special local active ferment, and quite independent of the amount and the condition of the uric acid found at other areas in the body, and he has renounced the view that the uric acid has a causal relationship to gout.

Haig's views stand alone in their originality and unhesitating boldness (Ewart). He believes that uric acid, or perhaps xanthin bodies, are the root evil of a great many disorders other than those generally accepted as gouty, and he bases his views on observations made on the acidity of the urine and the uric acid - urea ratios taken along with hypothetical conclusions as to the state of the blood and tissue juices. Personally I have never been able to obtain results which harmonise with his records, and he is to my mind unduly credulous in his interpretation of the state of the urine, blood, etc., as ascertained by the methods employed. The beneficial effects of the treatment advocated by him are undoubted in many cases, but the appropriate explanation is not, I think, to be put along the uric acid-urea lines referred to by him.

Sir William Roberts, 1892, as a result of an important series of chemical investigations on uric acid and its various combinations, advanced the view that there are three compounds of uric acid (H_2U). These are, the neutral urate, M_2U, where the metal replaces all the displaceable hydrogen; the biurate, MHU, where half the displaceable hydrogen is replaced by the metal; and the quadriurate, H_2U MHU, where one-fourth of the displaceable hydrogen of two molecules is replaced by the metal. The neutral urates do not exist in the body; the biurate is found in the form of biurate of soda in gouty concretions; and the quadriurate he regarded as the sole form in which uric acid exists in normal urine. He inferred that in the normal state uric acid is primarily taken up in the system as a quadriurate, that it circulates in the blood as such, and that it is finally voided in the urine in that form. He wrote as follows:—In perfect health the elimination of the quadriurate proceeds with sufficient speed and completeness to prevent any undue retention or any accumulation of it in the blood. But in the gouty state this tranquil process is interrupted, either from defective action of the kidneys, or from excessive introduction of urates into the circulation, and the quadriurate, circulating in a medium which is rich in sodium carbonate, gradually takes up an additional atom of base, and is thereby transformed with biurate. The biurate thus produced exists at first in the hydrated or gelatinous modification, but with the lapse of time and increasing accumulation it passes on into the almost anhydrous or crystalline modification, and the precipitation of it becomes imminent or actually takes place (Roberts).

He then made a series of investigations into the conditions which in an artificial parallel accelerate or retard the gradual transformation into the crystalline biurate, and concluded that sodium salts immediately hastened it. As a practical outcome of these observations he strongly deprecated the use of sodium salts, either in the form of medicines or as sodium chloride in the diet, and more recently Luff has recommended the use of salts of potassium in preference to sodium on somewhat similar grounds. Clinical evidence generally hardly supports this view. By way of illustration consider the very large quantities of sodium chloride taken by patients at Weisbaden, where the main springs are notoriously rich in common salt. And there is no doubt that in the usual run of cases equally good results are obtained by treatment with these waters as with others in which sodium chloride or other salts of soda are only present in very sparing amount. Notwithstanding Roberts' elaborate researches we must regard the view of the existence of a quadriurate, more especially in the blood, as a pure hypothesis and not as a proven fact, and it is probably unwise to lay any stress on experimental observations on the "maturation" of the uric acid compounds outside the body as furnishing a basis for any deduction in the way of therapeutics.

Ord in 1872 advanced the view that inflammatory or degenerative changes in the affected tissues are to be regarded as the primary cause of gout, such initial changes not being caused by urates. He believed that gout was the sequel of a special form of degeneration in some of the fibroid tissues, resulting in an excessive formation of urate of soda, which passes into the circulation, and is later deposited in areas least freely supplied with vessels and lymphatics.

Sir Dyce Duckworth is the leading exponent of the view which regards gout as intimately connected with a disordered state of the central nervous system. He believes that, as a result of a disorder of some part of the neurotrophic system, a derangement of metabolism is induced which leads to undue formation of uric acid, and also inhibits the normal breaking down of that substance in the tissues, these things being accompanied in the case of acute gout by temporary weakness in the excretory capacity of the kidneys.

Kolisch in 1895 suggested that some antecedents of uric acid are the cause of the toxic effects which he believes constitute the primary cause of gout. This theory was mainly based on the presence of an increase in the alloxur substances (xanthin, etc.) in the urine of the gouty, but in view of the results of numerous investigations recorded in the last few years showing no such increase, this idea is no longer maintained.

A study of these various views shows how little we really know of the etiology of the disease. Garrod's great discovery was unfortunately followed by the result that all attention has been focussed on uric acid as the all-important factor, while in reality there is every reason to believe that it is by no means the main etiological factor, but only one of the incidents in the development of the disease.

The writer has lately described the appearance in the blood in gout of certain peculiar forms of leucocyte, which were considerably increased in number during the paroxysm. Whatever the nature of these cells may be, their occurrence lends force to the view that we must take a broader and much more general view of the disease than heretofore, and not restrict our investigation to any single line of inquiry.

With our present knowledge it is futile to theorise as to the etiology of the disease. All we can do, or at any rate all we ought to do, is to indicate clearly the known facts as to the state of the urine, blood, and tissues in the disease, and wait for the further advances in our knowledge of physiology that are absolutely essential for the elucidation of the problems presented by the tissues in gout (see "Uric Acid").

Before passing to a consideration of these special points, reference must be made to certain general facts of some importance in the etiology of the disease.

Sex and Age, etc.—The disease in its most characteristic forms is more common among men than women, but its various irregular and minor manifestations, especially those seen in hereditary cases, are at least as common in the female sex. When there is a strong hereditary history, typical joint attacks may occur in young men in their teens and onwards, but as a rule such attacks are more frequently seen at and about the fourth decade. In hereditary cases various undoubted indications of a gouty tendency may be exhibited almost from infancy onwards; in other cases the first manifestation of the disease may develop in old age. With regard to bodily conformation it is usually supposed that persons of large frame and vigorous appetite, with a tendency to corpulence, are specially predisposed to gout. This is in the main true (such persons eat largely and often take little exercise), but at the same time many of the most typical gouty subjects are thin, pale, and dyspeptic-looking individuals.

Climate and Season.—Trousseau stated that one of his patients told him his joints were "barometers," and general clinical appearances at the present day afford not a few corroborative examples. The influence of meteorological conditions on general well-being (intra-cellular metabolism) is profound, but the nature of that influence is quite unknown. It has long been considered that gouty subjects fare better in an inland and hilly country than at the seaside, and there is a good deal of evidence in support. With regard to seasons, it is generally held that acute attacks of the disease occur with greatest frequency in the spring and autumn, but it is doubtful if this is really so to any noteworthy extent.

Drinking-Water.—It is probable that the drinking-water exercises a greater influence on gout than we know of at present. There is sufficient evidence to indicate that waters rich in lime are unsuitable, and there is little doubt that a combination of a clay soil and lime rich water are particularly injurious.

The Blood in Gout

Uric Acid.—The blood in gout contains an excess of uric acid. In well-marked cases, and especially during the paroxysm, this can readily be determined by Garrod's thread test, which is performed as follows:—To two drachms of serum obtained from a blister (applied at a site other than the inflamed spot) add ten to twelve drops of strong acetic acid. Mix the two fluids, and immerse one or two linen threads, and set aside for twenty-four hours. Then examine with a low power of the microscope, and if a positive result is attained, numerous minute rhombic crystals of uric acid will be found on the submerged part of the thread.

But the exact relationship between this increase of uric acid in the blood and the clinical features of the disease is by no means determined. One difficulty in determining this lies in the fact that uric acid is frequently present in considerable amount in the blood in conditions which have no known relationship to gout. Specially is this so in leucocythæmia, when there may be an amount of uric acid present in the blood far in excess of that seen in cases of gout, and yet there are present in these cases none of the clinical features either of acute, chronic, or irregular gout. Similarly with other morbid conditions, *e.g.* lead poisoning, although to a much less degree. The writer has recorded (*Brit. Med. Jour.* Jan. 28, 1899) the results of an investigation of the blood in cases of pneumonia, malignant disease, chronic Bright's disease, ulcerative endocarditis, and acute aneurysm, in all of which the presence of uric acid could be determined in the limited quantity of blood examined. Similar results have been found by other observers. Petren (*Archiv f. exp. Path. u. Pharm.* Bd. xli. 1898) found it present in a case of hysterical hæmatemesis and in one of gonorrhœal rheumatism, and he further emphasises its presence in anæmia as well as other diseases above mentioned. Weintraud also found that even in the healthy, uric acid can be demonstrated in the blood after the administration in very large amount of articles of diet rich in nucleins. These various points alone prove how inadequate the one factor of uric acid is in the etiology of the disease. Variations in the amount of uric acid in the blood clearly do not constitute the root evil, a fact emphasised by the point already noted, that Magnus Levy has shown that there is no appreciable difference in the amount of uric acid present in the blood during the acute attack compared to the free interval.

The Form in which the Uric Acid circulates.—This is in reality quite unknown. Sir W. Roberts' view has been already referred to, but as we know so little of the behaviour of the proteids, and also of the acid and alkaline salts of the blood in their relation to uric acid, we must regard his view as a hypothesis and not as a known fact.

Alkalinity of the Blood.—Much has been said and written about variations in the alkalinity of the blood being an important factor in the genesis of gouty symptoms. As with some other aspects of the disease, a good deal more is written on the subject than is warranted by facts. Reference has already been made to the importance of Magnus Levy's observations, as indicating that there is no appreciable difference in the reaction of the blood during the paroxysm compared to the free interval. Notwithstanding the writings of Haig and others, it may safely be asserted that there is no authoritative instance on record in which a diminution in the alkalinity of the blood has been clearly established (v. Noorden).

The Cellular Constituents.—The red blood corpuscles show no material change. The blood-plates exhibit no increase nor other definite abnormality, a point of some interest in connection with the undoubted liability to thrombosis in gouty cases. Balfour has indeed suggested that the phenomena of the acute attack may best be explained on the view of a local thrombosis. The only striking changes observed have been in the leucocytes. The writer has recorded (*Brit. Med. Jour.* Jan. 6, 1900) the appearance of a peculiar type of white cell, large in size (15 μ), with a large oval or horseshoe-shaped nucleus, poor in chromatin, the protoplasm vacuolated and imperfectly stained.[1] These cells could be readily differentiated from the ordinary finely granular oxyphile leucocyte, and also from the lymphocyte; in form and general appearance they resemble degenerated myelocytes. During the acute attack these cells were considerably increased in number, and their presence suggested the possibility of their having an important relationship to the alterations in the uric acid and phosphoric acid excretion observed in the same case.

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[1] In some cases the films were fixed with heat (120° for fifteen minutes), and in others with alcohol and ether for an hour. Some were stained with eosin (2 per cent watery solution for

The results of a more recent investigation, in course of publication, showed the presence of somewhat similar cells, and their increase in number during the paroxysm was even more striking than in the former instance. This case is possibly of even greater interest, as it is more than possible that the acute attack was in great part induced by the artificial administration of nucleic acid, which was tried as a therapeutic agent. Much further investigation is necessary before we can speak definitely as to the significance of these cells, and in the meantime we may refrain from theorising regarding them. Their presence in the two cases, and the great increase in number during the acute attack, are, however, suggestive of their possessing considerable etiological significance.

THE URINE IN GOUT

As we may look to the acute paroxysm to furnish us with the clue to the etiology of the disease, we will in the first place consider the condition of the urine during the acute attack. It is usually said that the urine then exhibits all the characters of a febrile urine, but this is by no means always the case. Transient albuminuria is present in a number of cases, especially in recurring paroxysms. The greatest interest, however, centres in the excretion of uric acid and other nitrogen-holding substances. There is now abundant evidence to show that during the attack there is not only no diminution in the amount of uric acid excreted, but there is frequently an actual increase, sometimes considerable. As already mentioned, Kolisch's view that during the paroxysm the alloxur bases are diminished has not been confirmed by other observers. We must also bear in mind that the laboratory difficulties in the investigation of the alloxur bases make their records less valuable than those of other substances.

Of greater importance is the excretion of total nitrogen. This is considerable during the attack, but this is doubtless in part due to the general febrile reaction. From the numerous detailed records available there is some evidence to show that for some time preceding the attack there is an actual retention of nitrogen in the tissues. The uric acid-urea ratio may be very little different from the normal, either during the attack or in the interval. With regard to the reaction of the urine very little can be said, except that we cannot lay stress on any apparent slight daily variations in its acidity. I have personally examined cases where the reaction was alkaline or neutral one day, and distinctly acid the next, and yet the amount of uric acid in the former was much less than that in the latter. Another point may be referred to. Attempts have lately been made to prove that the excretion of uric acid and phosphoric acid (two end products of nuclear disintegration) go hand in hand. It is very doubtful if this is the case. My own observations are strongly opposed to this view, and on other grounds one is inclined against it. It is true that uric acid and phosphoric acid are two end products of metabolism of the nucleins, but we must remember that the uric acid formed in the body is probably capable of further and

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two minutes) and hæmatoxylin (Hansen's solution, three minutes); others with eosin and methylene blue. In those fixed with heat special attention was paid to the granular leucocytes.

ready transformation into urea, while there is nothing to indicate that phosphoric acid can undergo any further change in the economy.

As regards the urine in chronic gout, it is generally believed that the uric acid excretion is small in amount in the intervals. This may be so in some cases, but it is by no means invariably so. There is considerable individuality in each urine, and some of the worst cases show a very large amount of uric acid in the free interval. The occasional occurrence of glycosuria, phosphaturia, and oxaluria has already been referred to.

CONCLUSIONS

Do these findings in the blood and urine lead to any conclusions as to the etiology of the disease? We must unhesitatingly answer in the negative. At the same time, we will act wisely by refraining to formulate theoretical deductions. The generally accepted belief that the primary development in gout is the heaping up and deposit of uric acid in the tissues must be regarded as quite inadequate in view of the points referred to under the state of the blood. The relationship which the increase of uric acid in the blood bears to the local deposit is entirely unknown. Is there any actual increased formation of uric acid in gout? This query at once raises another, Where is uric acid normally produced? As the latter question has not yet received a final answer, we must speak with diffidence on the former. The older views, which regarded uric acid as an intermediate product in the combustion of nitrogenous substances into urea, have long been abandoned. The observations of Kossel, Mares, Horbaczewski, and others, referred to under "Uric Acid," have in the last ten years thrown much additional light on the subject. As a result of their investigations, the theory was advanced that uric acid was derived from *nucleins in the cell nuclein*, and that the excretion of uric acid bore a close relationship to the number of leucocytes in the blood. This theory has not been borne out by further recent investigations. The view was then advanced that the uric acid arose from decomposition of the *nucleins of the food*, and much discussion still rages on this point. Others consider that it is exclusively derived from the organised nuclein of nuclear disintegration, and attribute to the nuclein of the food an indirect action only, through an acceleration of metabolism and cell disintegration. There is no doubt that the administration of a diet rich in nucleins, *e.g.* thymus gland (sweetbread), leads to a very considerable increase in the excretion of uric acid, but the manner of interpreting this result is by no means clear. Many things have to be carefully considered. To illustrate: it is well known that in addition to uric acid other nitrogen-holding substances—so-called alloxur or purin bases—are excreted as the result of nuclear disintegration, but as our methods of determining these substances can hardly yet be said to be thoroughly trustworthy, conclusions arrived at from these investigations are not wholly reliable. Further, there is sufficient evidence to indicate that the organism of man can further transpose uric acid into urea, a fact of some importance in connection with views recently advanced as to the excretion of uric acid and phosphoric acid running a parallel course. These two substances are two end products of nuclear disintegration, and as there is no reason to believe that phosphoric acid undergoes any further change in the organism, while there is good reason to believe that uric acid can do so, it would be surprising if the excretion of the two substances went strictly hand in hand. Apart from this theoretical reasoning, my own investigations have convinced me that no such parallel exists. There is also no doubt that the *nucleins present in cell nuclein generally* are a source of uric acid production, but the conditions which influence the formation and decomposition of these nucleins in the body are quite unknown. Other writers maintain that individual dispositions vary considerably with regard to the amount of uric acid produced under apparently similar conditions. The whole subject of uric acid formation is still obscure, and it may well be, as Minkowski suggests, that the significance of nuclein-holding articles of diet may ultimately fall quite into the background. We must remember that the experiments recorded with thymus feeding cannot be regarded as comparable to the ordinary dietetic measures in daily use.

We are not in a position to make precise statements as to the conditions which influence the formation and excretion of uric acid in health, and still less are we qualified to speak of the state of affairs in gout. This being so, it is not surprising to find that the fundamental questions in the problem of gout are variously answered. One such question pertains to the kidneys. Are the kidneys primarily or mainly at fault in gout? This may be so; but if so, this has not to do with uric acid. Not only is the actual output of uric acid not diminished (often actually increased) during the acute paroxysm, but I have lately shown that the tissues of a gouty subject react like the normal tissues to the influence of pure nucleic acid (*Jour. of Path.* 1900), and Knoll had previously shown that they react like normal tissues to the influence of thymus feeding. In gout it is yet impossible to state whether we are dealing with an increased formation of uric acid, a diminished excretion, a diminished oxidation, an alteration in the solubility, or other unknown factors. Much further investigation is necessary before these points can be determined.

Finally, I will merely throw out a few suggestions which occur to me as having some importance in connection with the etiology. These remarks or queries will be based mainly on the following two points: (*a*) There is ample evidence to prove that the uric acid in the blood is not the primary factor in gout; and (*b*) uric acid can be deposited in cartilages and other tissues, even in considerable amount, without the association of any inflammatory phenomena.

(i.) The last-mentioned point clearly proves that the uric acid is not the factor which causes the inflammatory phenomena characteristic of the acute attack.

(ii.) What, then, are the toxic principles in the blood which possess the power of inducing the characteristic inflammation? and

(iii.) What are the factors which determine the incidence of these toxic substances from the blood into the tissues?

In connection with these queries we have to consider the all-important part played by the alimentary canal. Here we have, doubtless, one of the important keys to the solution of the problem.

(iv.) What effect has the absorption of the products of abnormal digestion of proteids on the blood? This abnormal digestion may arise from quite a number of circumstances, *e.g.* excess of proteid, faulty admixture of food stuffs, the effects of alcoholic stimulants, the result of fatigue from excessive muscular exercise, etc. In this connection it is well to emphasise that this question will never be ascertained by mere analysis of the intestinal contents. What we desire to know is not so much the nature of the toxines as the reaction of the tissues—blood, blood glands, and tissues generally—to their influence. Yet another query.

(v.) Are the injurious effects of various carbohydrates, liquors, and other substances to be explained solely by their interference with the digestion of the proteids in the alimentary canal, or have they in addition the power of more directly exercising an injurious influence on the blood and tissues ?

HEREDITY

The tendency to gout is inherited; it is not infrequently transmitted by the female line (cf. Chart), although specially manifested in males; in hereditary descent the tendency becomes inextricably mixed up with the tendency to other diseases—rheumatism, rheumatoid arthritis, tuberculosis, etc. If we add to these points the statement that environment, using that term in its widest sense, is an all-important factor in the explanation of the numerous peculiarities seen in hereditary transmission, we have stated all that is definitely known on the subject. Hutchinson and Duckworth accept the French view of the existence of an arthritic diathesis, from which arise as branches two distinct classes of disorders commonly recognised as gout and rheumatism. The former further agrees with the views of the older writers who recognised a mixed condition of the two disorders. There is every reason to believe that this mixed condition frequently occurs, but the term rheumatic gout is a misleading and inaccurate one. I do not intend to enter into any discussion as to the relationship of gout to rheumatism and rheumatoid arthritis, but refer the reader to the valuable writings of Hutchinson and Duckworth. While the subject is an interesting and fascinating one, our present knowledge will not admit of any practical observations on it. As Thomson has well said, the hopeful outlook is not in theorising but in experiment, in the collection of precisely observed data, and in the skilful use of statistical methods. .

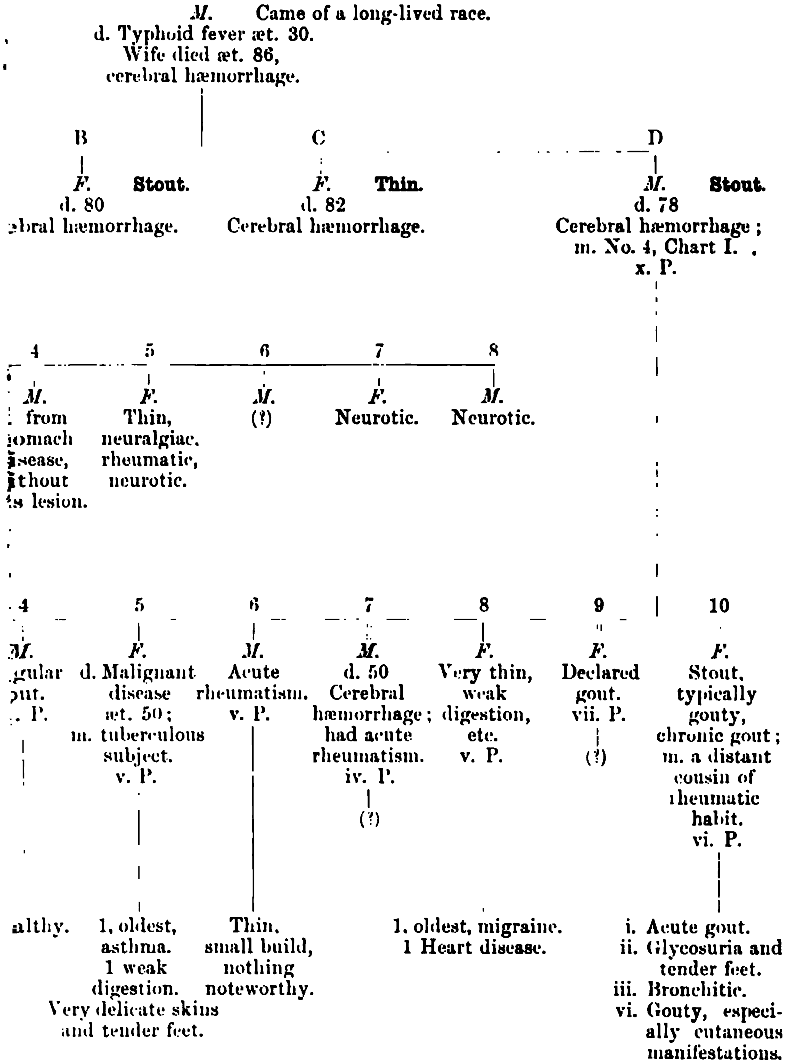
In gout certain experimental investigations are necessary before our knowledge of its hereditary aspects can be advanced. These can best be carried out by the induction of gout in lower animals, *e.g.* hens, etc., observing the effects of transmission of the gouty tendency to succeeding generations. At the same time, the statistical method is of undoubted value, but from its nature is clearly less valuable unless the statistics are complete and reliable, a combination not readily obtainable in practice. For some years I have been interested in carrying out this method as opportunity afforded, and have been fortunate in securing a particularly good illustration of the effects of the transmission of the disease. I am satisfied that the information given in the charts is reliable, but it is doubtless more incomplete than indicated. Nevertheless many points of the greatest interest can be learned from them. The relationship to rheumatism, diabetes, obesity, tuberculosis, malignant and other diseases, all find representation in a single family.

Gout is a general disorder of metabolism, and its adequate consideration requires careful study of the various derangements seen in succeeding generations, which in turn includes a consideration of variations in environment in the different individuals. With regard to one of the associated derangements of metabolism it is interesting to note that according to the Registrar-General's returns the mortality from diabetes is on the increase ("Diabetes," vol. ii.).

DIAGNOSIS AND PROGNOSIS

The diagnosis of gout must be made from the history and general character of each case. In acute gout, and in chronic cases with well-marked external manifestations, there is, as a rule, no difficulty, but in many other cases there is no pathognomonic symptom whatever to distinguish it from

CHART III.


M. Came of a long-lived race.
d. Typhoid fever æt. 30.
Wife died æt. 86,
cerebral hæmorrhage.
B
F. Stout.
d. 80
ebral hæmorrhage.
C
F. Thin.
d. 82
Cerebral hæmorrhage.
D
M. Stout.
d. 78
Cerebral hæmorrhage;
m. No. 4, Chart I.
x. P.
4
M.
from
omach
sease,
thout
s lesion.
5
F.
Thin,
neuralgiac,
rheumatic,
neurotic.
6
M.
(?)
7
F.
Neurotic.
8
M.
Neurotic.
4
M.
gular
out.
. P.
5
F.
d. Malignant
disease
æt. 50;
m. tuberculous
subject.
v. P.
6
M.
Acute
rheumatism.
v. P.
7
M.
d. 50
Cerebral
hæmorrhage;
had acute
rheumatism.
iv. P.
(?)
8
F.
Very thin,
weak
digestion,
etc.
v. P.
9
F.
Declared
gout.
vii. P.
(?)
10
F.
Stout,
typically
gouty,
chronic gout;
m. a distant
cousin of
rheumatic
habit.
vi. P.
althy.
1, oldest,
asthma.
1 weak
digestion.
Very delicate skins
and tender feet.
Thin,
small build,
nothing
noteworthy.
1, oldest, migraine.
1 Heart disease.
i. Acute gout.
ii. Glycosuria and
tender feet.
iii. Bronchitic.
vi. Gouty, especially cutaneous
manifestations.

NOTES.

to the very frequent presence of urate of soda in the joints of those patients who die of
and D are of considerable interest. Their longevity is noteworthy, and so also are the two

shows the great influence of the mother on the progeny. The neurotic strain is the pre-
ing from some gastro-intestinal neurosis, a pathological examination revealing no gross
l themselves as rheumatic.
original tendency to longevity, observe the apparent effect of admixture with a known gouty
t an average of 56; note the history of each.
t bodily conformation, noted in a previous generation, are very marked—three are very thin,
. The stout members have a double line.
all the families of the same generation marked with an asterisk. These are the children of

illustrating a blend with tubercle. In the former case the subject was not *herself* tuber-
lied from tuberculosis; in the latter the patient was *himself* tuberculous. The history of
the similar ones in Chart II. In the cases under consideration the comfortable circumstances
l to foster a gouty tendency. Taken collectively they illustrate the all-important influence of

of a rheumatic and gouty strain—an intensification of the gouty tendency.

rheumatism, with which it is liable to be confounded. The intermediate diagnosis of the two diseases approach each other so nearly, that to discriminate between them with our present knowledge may be impossible. In acute gout of the monarticular type a differential diagnosis has to be made from gonorrhœal arthritis. At least one case of very pronounced gout has come under my observation where the initial attack, which occurred at the age of twenty, and which involved the right wrist and back of hand, was erroneously regarded as a gonorrhœal arthritis. In this connection it may be well to state that Jonathan Hutchinson believes that gonorrhœal arthritis is more prone to occur in people of gouty habit, but in this instance that origin could be reasonably excluded, and the great rarity of the condition in a Scottish hospital fairly explains the error in diagnosis. In a few cases of acute polyarticular gout it may be by no means easy to differentiate acute rheumatism, and careful regard must be paid to the state of the blood, the previous history, and the measure of reaction to the remedies appropriate to each disease. Salicylate of soda in sufficient doses will certainly influence acute rheumatism, and will probably have very little or no effect on acute gout, while, on the other hand, colchicum will almost certainly exercise a remedial influence on the latter disease.

Although much could be written on the subject of diagnosis, much uncertainty must still attach to many instances of irregular gout, *e.g.*, lumbago, sciatica, and the like. After all, the distinction is often of little practical importance, as these cases are usually adults or older subjects, in whom the treatment of the individual case will not differ markedly to whichever genus of disease it may be assigned. (See also "Rheumatism.")

In a young subject affected with inflammatory or allied conditions in the hands or feet, or other irregular manifestations, especially if there be a distinct hereditary history, the possibility of the gouty nature of the affection should always be kept in mind, and while we must avoid the custom of attributing every morbid action occurring in a gouty habit to the influence of gout, we will be well advised in keeping in view the possibility of such a relationship.

Prognosis.—The prognosis of gout depends on so many circumstances, that it is hardly possible to speak with any precision on the point. Much depends on the age of the patient, the duration of the disease, the presence of complications, especially the state of the kidneys and cardio-vascular system, and on the patient's will power and determination to give full effect to all appropriate treatment. Each case must be judged on its own merits, and if all these things are equal, which they seldom are, the prognosis, both as regards life and recurrence of the attacks, is eminently favourable. But we must admit that in a very small proportion of cases, in whom the disease develops early, and who give fairly full effect to appropriate treatment, there are a few apparently inexplicable recurrences of the disease. Such cases are, however, very exceptional; and the guarded prognosis, which is usually advisable, largely depends on the fact that the establishment of a rational and efficient prophylaxis is, from the nature of things, by no means readily attained. The prognosis from the point of view of life insurance will be referred to under that heading.

TREATMENT

General Considerations.—As the exact nature of the primary changes which lead to the uratic deposits characteristic of the fully developed disease is unknown, in the treatment we must be content with the adoption of those general and special measures which experience has proved to be beneficial in arresting the frequency and severity of their occurrence. These measures vary within wide limits. In the pre-uric-acid days the older writers regarded gout and plethora as practically synonymous, and they recognised two different types: one, the plethora of a healthy and vigorous habit, which is readily acknowledged and seldom misunderstood; the other, arising in a constitution more weakly by nature or depraved by vitiating influences, is characterised by gouty phenomena which are more liable to mislead. These clinical types still exist, and their careful study and appreciation will assist in arriving at the treatment appropriate in different cases. These remarks hold good for the fully-developed disease, but, as we have seen, we are much more frequently confronted with the treatment of minor and irregular symptoms, which develop early or late in life, and persist or frequently recur without ever culminating in the grosser manifestations. This, the early stage of the disease, is of the first importance. It is calculated to throw light on all the subsequent changes, and has neither received the attention to which it is eminently entitled, nor has it been given the place which it ought to have both in determining the true pathology of the disease and in establishing the treatment best suited to its relief (*vide* Mr. A., p. 242). In fine, the treatment of gout and the gouty diathesis is essentially a prophylactic one, and the results will depend on the length of time and the care with which the necessary prophylactic measures are carried out.

Of the treatment to be recommended and described, general measures relating to an all-round judicious mode of life are of much greater importance than any special medicinal or other remedies. A victim of gout soon learns that there is no radical remedy for his disease, and he sensibly holds himself aloof from all useless interference, relying more on careful regulation of his life, diet, and excretions. In arriving at a knowledge of the appropriate treatment, due regard must be paid to any peculiarity of constitution, whether natural or acquired, which may vary the aspect of the disease, and, further, the greatest assistance may be obtained from an observant patient, the facts so gained furnishing an important clue to the line of treatment to be followed in any given case.

While it is by no means determined that an increased uric-acid formation is a cause of the disease, there are sufficient indications to warrant us regarding a *diminution in the production of uric acid* as an important indication in treatment. This can best be obtained by, in the first place, regulating, *i.e.*, diminishing, the total quantity of food consumed. The fact that through immoderate eating and drinking gout is favoured cannot be gainsaid, and while it is true that the disease may develop in people who are poorly and inadequately nourished, this fact merely proves that other influences may induce a like result. We must clearly distinguish between absolute and relative excess of food. By the former is meant that which would be excessive in the most healthy state of the individual constitution; by the latter we mean that which, although it may not exceed what the individual in a state of health, with moderate exercise, might safely indulge in, is yet relatively excessive when health has from any cause deteriorated. So long as the excesses are casual and inconsiderable, the self-adjusting powers of the body are sufficient to dispose of them; but when, from extent or continuance, the excesses strain these powers beyond a certain point, the corrective energies of nature fail, and the outward and visible evidences of the disease result. In such cases well-marked constitutional disturbance had existed without the individual having any consciousness of its presence, which fact indicates that a decision as to the state of health of a typically gouty subject cannot be arrived at from the evidence of his own consciousness. There is also sufficient evidence to justify *a limitation in the articles of diet rich in nucleins, e.g.*, thymus, liver, kidneys, but, as these form a very small part of ordinary feeding, they can, except in special cases, be overlooked.

If these points are attended to, a further indication in treatment is at the same time fulfilled, *i.e., the elimination of the uric acid.* It is well known that defects in the renal functions play an important part in the disease, and, if the strain on the kidney is lessened through administration for a short or long period of a reduced nitrogenous dietary, these organs will be enabled to cope more successfully with the elimination of uric acid and other toxic substances. This object will, of course, be more readily obtained if the assistance of the correlated excretory organs, the skin and intestinal canal, is secured and maintained.

Allusion has already been made to the older views on plethora, especially that arising from excess of nutriment, so-called nutritive plethora, the treatment of which mainly resolved itself into a limitation of the amount of food consumed. But the older physicians also recognised an "excrementitious plethora," usually a sequel of the other, with resulting far-reaching injurious effects on all the organs and tissues of the body. This condition suggested to them the advisability of closer attention being directed to the excretory function of the skin by means of baths of various kinds. Since that time Balneology has developed extensively, and has rightly come to occupy a very important place in the therapeutics of the disease. The action of baths in general on the heat-regulating function, on local and general tissue metabolism, on the general circulation, and on the central nervous system, are elsewhere described (*v.* Balneology), and need not be considered in detail. It must suffice to indicate the very important part which the skin can play as an organ of excretion of nitrogenous waste products (urea). This fact, of which there is abundant clinical evidence, is one which is insufficiently appreciated in most text-books on physiology, and the practical deductions to be drawn from it are too much ignored by the practitioner.

An interesting question relates to the subject of *uric acid solvents.* If we can so influence the blood and tissue fluids as to increase the solubility of the uric acid compounds, and thus facilitate their elimination, it is obvious that this will be one of the outstanding indications in treatment. As on many other points, opinions vary both as to the theoretical and practical value of these substances, but it is on the whole doubtful, if by the administration of any alkaline salts, the remarkably stable blood and tissue fluids can be so influenced as to lead to their exercising a solvent action on the deposit. In the absence of further proof, it is probably safer to conclude that any beneficial action possessed by these substances is primarily due to their diuretic action. The same statement can be applied to the use of the various organic nitrogen-holding bases—piperazin, lysidin, urotropin, etc.

A point of greater importance, and of even greater difficulty, is the influence of the preformed salts in the different articles of food on the solubility of the uric acid. Here, again, we are on unknown ground, and if we are not in a position to speak definitely regarding the influence of the preformed salts of the animal and vegetable foods, still less are we able to speak of the action of the salts formed in the body from oxidation processes. The sulphur and phosphorus of the albuminous food-stuffs are oxidised into their respective acids; these cannot exist as such, and are immediately changed into various compounds; but the nature of these changes, either in healthy or deranged metabolism, is not determined. Similarly, while the organic acid salts present in vegetables are changed into alkaline carbonates, we do not know what influence they exert on uric acid compounds in the blood or tissues, and it is probably inadvisable to draw any conclusions from the solvent power of these and other substances *in vitro* applicable to the state of affairs in the living body.

We are on safer ground when we pass to consider *the beneficial influence of exercise* on the gouty subject. Everything which favours the inner or tissue respiration in the muscular and other tissues directly or indirectly fulfils the preceding indications, and in this category work and physical exercises of different kinds take a first place. These exercises should preferably be conducted in the open air, and should be of a kind adapted to the age and sex of the individual subject. Active walking, golfing, cycling, horse exercise, hill-climbing, and other seasonable exercises occupy a foremost place in the list. With regard to the extent to which these are to be advocated no hard and fast line can be drawn; the habit of body of each individual, and the state of the cardio-vascular system, must be carefully considered in each. Needless to say, the presence of organic disease in the kidneys, heart, or other organ will modify the extent to which these are to be indulged in.

While it must not be forgotten that in a few cases active exercise seems to have a prejudicial influence in inducing an acute attack, this should in no way interfere with the general application of this principle. More harm results from too little than from too much exercise, and we should remember that the name of exercise is often given to bodily exertion so gentle that no increased activity of inner respiration results. When time and opportunities do not allow sufficient exercise in the open air, a fair substitute is found in the various available forms of home gymnastics, and in many cases a judicious combination of the two is very desirable. In all cases attention should be directed to those groups of muscles which, from the patient's occupation or disposition, are apt to be largely or entirely neglected.

Still more active measures are frequently advisable in cases with chronic joint affections, or in the case of involvement of fascia or local muscular and connective tissues. Such cases may be much benefited by various forms of active and passive movements (*vide* Medical Gymnastics). The benefit to be derived from this theraputic measure may be emphasised by the fact that not a few typically gouty (usually irregular gout) adult subjects, men and women, regularly undergo, of their own accord, a course of medical gymnastics in one of the numerous Swedish mechanical institutes available for this purpose at the various foreign spas.

A further general consideration requiring attention is the *state of the central nervous system.* While the importance of this factor in this, as in most other diseases, is undoubted, there is, I think, a tendency to lay undue stress on it. While it is true that recurring indications of declared gout not frequently appear to be dependent on nervous causes, in so far as its manifestations declare themselves after some mental perturbation, typical attacks of acute gout occur with sufficient frequency, both in elderly and younger subjects, quite independently of any such exciting cause to justify us not laying very great stress on the relationship. Further, most of the undoubtedly gouty developments in young subjects arise and recur apparently independent of any direct nervous influence. On this ground, also, we are led not to lay very great stress on the nervous system as *the factor* of primary importance. Its ultimate importance cannot be gainsaid. It is one of the factors which continually varies the aspects of the disease.

Derangements in the digestive system play an exceedingly important rôle in the development both of acute and chronic gout, and these derangements usually depend either on immoderate eating or drinking, or on the ingestion of articles of diet especially unsuited to the individual. The probable relationship of these digestive disturbances to the phenomena of acute and chronic gout has already been referred to, but be that what it may, the importance of a well-regulated and healthy intestinal mucous membrane cannot be over-estimated. Auto-intoxication is certainly a primary factor in the disease, and has to be guarded against by careful dieting, healthy intestinal secretion, and normal intestinal evacuation. There is no doubt that in a certain proportion of cases a hydragogue cathartic, taken on the first indication of impending trouble, will avert the acute attack, and it is advisable to insist at all times on the closest attention being paid to the action of the bowels. While we have yet to learn much about the secretory or excretory influence of the large intestine, we may safely assume that by judicious purgation we do much more than merely free the system of the undigested food-stuffs present in the gut. The vigour of the constitution must be the standard by which the depletion is regulated, and where such vigour is deficient, measures of the same activity should not be pursued as are necessary when the system is robust. Not infrequently in the course of subacute or chronic gout there is developed a subacute or chronic catarrh of the gastric and intestinal mucosa, which in turn aggravates and complicates the general gouty state. Such a complication merely leads to a more strict application of the general principles of the dietetic treatment appropriate to the disease.

Dietetic Treatment

It is sometimes maintained that the influence of different food ingredients on the gouty constitution has been too much magnified, this assertion being based on two clinical facts—firstly, that a child fed entirely on milk may continue to excrete large quantities of uric acid; and, secondly, that an adult patient who has been excreting a similar excess on a light vegetarian diet may speedily improve and return to a normal excretion when his diet is changed to meat. This view is based on too narrow a conception of the problems. The little we know with certainty regarding gout seems to indicate that the radical defect is in the metabolism of the proteids of the food. The metabolic changes of the proteids of milk or vegetables are essentially similar to those in meat, and we have no reason to believe that the decomposition products arising from the normal metabolism of the one are in any way different from those in the other. Any differences there may be are those of degree and not of kind. Everything depends on the form in which the food-stuffs are presented. It sometimes strikes me that in this question of feeding we find an interesting analogy in the art of agriculture. A scientific farmer in the feeding of his land has not only to consider the natural quality of the soil, but also the climate, rainfall, and nature of the product desired. Of the food required by the growing plant the element nitrogen is also one of the mainstays, and it is usually provided in one of two forms—potassium nitrate or ammonium sulphate. Now it is by no means immaterial to him in which of these forms the land is fed. His choice depends on many things, one of the most important from our present point of view being their relative solubility. If the land is badly drained, and especially if the rainfall be considerable, he uses the less soluble ammonium sulphate in order to minimise the risk of the nitrogen being washed away and so rendered unavailable for the growth and maintenance of his grain. So it is with the human subject, but only in a more elaborate way. The individual qualities of the tissues, their drainage system, and the relative solubilities of the different food ingredients as commonly prepared, constantly demand careful consideration. The question of diet is certainly the paramount one. Its influence is profound, not only on the individual, but on the race, and its importance was well defined by Sir William Roberts when he wrote that one generation of scientific dietetics would produce an influence upon humanity second only to a new creation of the race.

MEATS, ETC.—From the earliest times some writers have regarded all or at any rate most kinds of muscle food as injurious to the gouty, and as in other debated points in the subject history of the disease many experimental observations have been brought forward to prove this point, but, as a rule, these observations have been made in the much too narrow field of uric acid excretions. The most recent observations (Taylor) indicate that the commonly accepted view that a meat diet is associated with an increase in the uric acid excretion is an erroneous one, and on the whole we may take it as definitely shown, both by practical experience and theoretical experiments, that a gouty subject may take a measured quantity of meats in an easily digested form.

In the use of meats it is not only important that these should be taken in an easily assimilable form, but that they should not be accompanied by an undue admixture of other food-stuffs. It is held by some that in such cases the fact of the carbohydrates and fats being more readily oxidised in the tissues, leads to defective combustion of the albuminous foods. This subject has been previously discussed, but whatever the scientific truth may be, the fact remains that we must look for the cause of any injurious effects of meat more in its quality and in the form in which it is administered. If too little nitrogenous food be taken an increased decomposition of nuclear-holding tissues may result, as has been proved in a case of complete starvation, when typical acute gout developed in the course of the observation.

We must, however, bear in mind that a strong meat diet, that is, meat twice or thrice daily, is an acid food owing to the imperfect neutralising of the sulphuric and phosphoric acid previously referred to. This may be in part rectified by the consumption of the alkaline table waters referred to later.

We have also to consider that the tastes and inclinations of the greater number of gouty subjects demand a certain supply of meat, and, by taking it in moderate amount, the supply of nitrogenous food necessary for the maintenance of the albumin in the body is more readily obtained.

With regard to the different kinds of animal food, white meats, *e.g.* fish and chicken, are more suitable than red meats owing to their more ready digestibility, and also in the case of fish to the smaller proportion of nitrogen present in equal bulk. The confirmed gouty subject is wise to limit his consumption of red meat to one meal in the day or even less, and further, to make as a routine a selection of the red and white meats similar to that indicated in case Mrs. E., p. 247. The whole question of a meat diet is summed up in its digestibility, which in turn bears a definite ratio to the simplicity of the meal in which the meat is a component part. The temporary diet of meat and hot water which is of such value in suitable cases, is a simple one, and to its simplicity we must largely look for an explanation of its beneficial effects. What has been said of meat holds also good for other animal foods. The various kinds of fishes and game can all be taken by a gouty subject, but what we must have regard to is the amount of admixture with other foods and drinks. Not infrequently innocent substances taken at the end of a highly nitrogenous mixed meal are regarded as the noxious agents when in reality they have played a quite subordinate rôle. High game and very fatty meats should be avoided.

Under this heading a few practical points may be mentioned about the culinary aspects of soups, meats, and fish. It is impossible to over-estimate the importance of this subject; the want of recognition of its importance is, I think, one of the causes of the very diverse differences of opinion entertained as to the beneficial or noxious influence of various dietetic substances.

Soups.—What soups may a gouty person partake of?

The answer to this question will depend on the digestive capacity, but in coming to a decision we must take into consideration some elementary points in the preparation of the various soups.

It has been stated that there are perhaps not less than 500 soups, but regarded analytically, there are only a few leading species from which the different varieties are produced by additions and combinations of flavours.

(*a*) A clear decoction of meat or bones which, when weak, forms a broth or a "tea," and when strong is a consommé or essence. These may be prepared from beef, veal, or mutton, and sometimes pork or ham.

(*b*) A similar decoction can be made from the various forms of fowl, game, and fish.

(*c*) A decoction of vegetables, including herbs, roots, grains, and farinaceous substances.

All of these soups may have added to them well-known dried Italian pastes, *e.g.* vermicelli or macaroni, and a consommé of meat or pork or game may be thickened with additions of a meat, fowl, or game purée respectively. In the same way the weaker broths furnish a basis for vegetable purées.

The following highly nitrogenous soups are not as a rule suitable articles of diet for the gouty:—Turtle, mock turtle, hare, kidney, oxtail, mullagatawny. If they are indulged in, the rest of the food and drink consumed at the same meal must be more carefully limited.

Cock-a-leekie, giblet, and hotch-potch are almost stews, and should be considered as a meat course. In not a few cases all of these highly nitrogenous soups are contraindicated.

The great fault to be found with soups usually served is that they are heavy, and contain too many ingredients. The average soup is made up with as many good things as possible, some to make it more nourishing, others to make it more palatable. This is all very well for the healthy, but where, as in gout, the digestive functions in the tissues and alimentary canal are readily disturbed, simple soups are required. The soups without thickening are therefore the most suitable. The thickenings in common use are purées of meat or fowl, flour, tapioca, yolks of eggs, etc.

A good rule in making soup is always to make it the day before it is required, as it has then time to cool, and the fat having risen to the surface can be easily and thoroughly removed. Soup should never be allowed to cool in the saucepan in which it has been made, but should always be poured into a basin, as otherwise the temptation to reheat without removing the fat is too great for most cooks.

Roast beef and mutton bones boiled with vegetables, and the fat carefully removed, make a good stock from which soup can be made for the gouty. Excellent soup can also be made from the water in which meat or fish has been boiled.

The various vegetable purées—spinach, artichoke, tomato, carrots, green peas, etc.—are excellent for this class of patient. They are sufficiently sustaining to prevent a feeling of hunger, and if well digested give a fair amount of nourishment. In the case of soups made from the pulses, their high nutritive value should influence the rest of the meal, and in some cases they are better avoided. A very good vegetarian stock can be made by extracting the "goodness" and flavouring from vegetables, the chief ones being onion, celery, carrot, and turnips. To do this take these vegetables and cut them into small pieces, place in a saucepan with sufficient water to cover them, and let them boil gently for several hours. The liquor when strained off is "stock," and can be flavoured with a small quantity of savoury herbs, pepper, salt, and ketchup, and can be coloured a nice brown with a few drops of Parisian essence.

A more detailed account of the methods of preparing the invalid soups (teas and essences) will be found under the heading Invalid Feeding.

Meats.—Made-up meats are not suitable for the gouty, owing to the greater toughness of fibres induced by the second cooking, and also because of the admixture of rich sauces of various kinds which are usually added for palatability. Meats should be tender and simply prepared. The best ways of preparing are in order—broiling, steaming, roasting, boiling, baking, stewing and frying. The last mentioned should be avoided, especially in the case of beef and mutton. Although lamb and veal possess less extractive value than other meats, and are on that ground commendable, yet the gelatinous nature of the fibres makes them more difficult of mastication, and therefore less digestive. If allowed, this danger must be pointed out with the view of obviating it. Tripe, sweetbreads, kidney, and liver may all be allowed for occasional use, provided the very special cleaning and careful cooking necessary are given effect to. Salted meats are rendered more indigestible in the preparation, and should therefore be avoided. Bacon and ham are more digestible than pork. With regard to game, white flesh is more suitable than brown, and water-birds are more fatty than other game.

Fish.—Fish are well calculated to form a large proportion of the dietary of the gouty. They contain on an average one-third less nitrogen than an equivalent amount of ordinary meat, and usually contain little or no fat. Fat fishes (salmon, mackerel, eels, pilchards, red mullets) are equal in nitrogenous value to an equal amount of moderate fat beef. There is a very large proportion of water in the flesh of fish. When ordering, it is well to suggest those known to be in season and plentiful, as they will probably be in the best condition. As has been already mentioned, many nutritious soups can be made from them.

Whiting, smelt, and sole are the most delicate and easy of digestion. Haddock, flounder, gray mullet, and plaice, are also good; the haddock has more flavour, but a coarser grain than the whiting. Mullet and halibut have firmer fibre, and are better cooked in large pieces. The fatty fish referred to are very tasty, but are more prone to derange digestion. In the salmon the fat is mainly found on the underside, and a slice from the back may be readily taken when the underside disagrees. Cod, unless in the very best of condition, is the least digestible of all white fish owing to the great toughness of its fibres, but the head, when boiled, makes an excellent stock for soup. Of the shell-fish, oysters, mussels, and scallop are all good, and so are also lobsters, crayfish, shrimp, crab, and prawn. Lobster salad or crab pie is quite allowable for young subjects with good digestion. Its nutritive value is not high, and its acceptability to the palate is beyond question. Fish should never be over-cooked. The best methods of preparation are boiling and steaming, then comes broiling or grilling, and lastly frying. In not a few cases we find fish are not permissible. Much, however, depends on the cuisinère. A combination of curried fish and rice makes a good dish. When digestion is obviously weak sauces are better avoided. One of the simplest is a little melted butter with chopped parsley. The familiar "butter sauce" is usually an imperfectly cooked indigestible compound.

Meat and Hot Water Cure.—Before leaving the subject of meat reference may be made to the value of an exclusively red meat diet in certain cases of chronic gout. Armstrong (*Med. Soc. Transactions*, 1897) has recorded a series of interesting cases, in which after a course of the usual routine treatment had failed, very great benefit was obtained by the so-called "Salisbury" treatment. The essentials of this treatment are the drinking of three to four pints of hot water daily, a pint to be taken before each meal, and the same quantity at bedtime, and the administration of one to five pounds of meat in the twenty-four hours. The meat should consist of beef-steak freed from fat, gristle, and connective tissue, well minced, a little water being added, and then warmed through with gentle heat. I have had no personal experience with this treatment, but can readily accept the moderate claims made for it in the paper referred to. The probable explanations of its value have been previously discussed. I would just like to add from experience in other cases that if the meat as thus prepared is not readily digested, a yet simpler and more digestible form may be found by taking the best beef-steak, freed from fat, and rubbing through a fine sieve until the juice has all passed through, and all the fibrous parts remain. The mince should then be treated as above. The appearance when cooked is just like very fine mince. The value of this preparation is by no means confined to gouty dyspeptics. The course of treatment lasts from four to twelve weeks, and thereafter the patient makes a gradual return to an ordinary dietary. The conclusions formulated by Armstrong from his experience of the treatment are as follows:—

1. That a certain number of cases (not more than 3 per cent) of chronic gouty

arthritis, recurrent uric acid calculi, and gouty dyspepsia, with fermentative changes, which have proved refractory to ordinary methods of treatment and dietary, may be treated by means of an exclusively red meat dietary, plus hot water drinking, with excellent results.

2. That this method of treatment is irksome and trying, and as, unless it is carried out *strictly* in the first instance, it is apt to do harm, it should only be used in those cases where other methods have failed, or are thought likely to do so.

3. That the cases require careful selection and close medical supervision, the details being modified according to the needs of each individual patient.

4. That those who suffer from persistent albuminuria or organic heart disease are in most instances unfit for this treatment; when, however, it is prescribed for them its course should be watched daily.

5. That certain cases of chronic gouty arthritis which fail to improve while on a mixed diet, recover equally well whether on this dietary or on the meat free dietary suggested by Dr. Haig.

6. That it is of the utmost importance that no addition, however small, of carbohydrates, saccharine matters, or fruit be made to the dietary during the first few weeks of treatment—very slight acts of carelessness in this respect having often caused disappointment and failure.

7. That used with due care and discretion this method is a most efficient and sometimes even a brilliant addition to our therapeutic measures.

The reader is referred to the original paper for a more detailed account of the treatment and of the reaction of the tissues under its influence.

MILK AND MILK PRODUCTS.—As in the case of various other foods, there is much difference of opinion as to the value or necessity of a diet composed largely of milk, milk products, and vegetables. In many cases a course of a strict milk diet is the most suitable, particularly in young and otherwise healthy subjects, but it is much less suitable for adults and the aged. Milk is highly nutritious, and when it is easily digested and no undue fermentation processes induced by its use, a limited course of milk diet is to be commended, the amount and duration being regulated by the effects on the digestive system and by the attitude of the patient towards it. Alkalinity of the urine is favoured or increased by a full milk diet, yet an exclusively milk regime is probably in the main unfavourable. However, there is no doubt that the children of gouty parents should be brought up systematically on a diet in which milk and its products are the staple, and meat given in very limited quantities. This is specially important in those by no means infrequent cases where there is, in addition, a marked neurotic strain in the family (*vide* "Adolescent Insanity"). With the active and fixed habits of later life a meat-free diet is very rarely practicable, and is very seldom called for.

Cream, forming as it does the most appropriate form of fatty food in the dietary of the aged, likewise constitutes an excellent form of fat administration in gouty subjects. It should preferably be taken with milk pudding or stewed fruit in an otherwise simple meal, or it may be used in the preparation of chicken cream, fish cream, or in various combinations with vegetables, when it takes the place of butter (*vide* "Invalid Cookery"). Cream which has not been treated by any artificial process is always to be commended.

Skim-milk is more digestible than ordinary milk in all cases where fat is not readily digested, but in recommending it as a beverage or food regard must be paid to the amount of proteids and lactose present in it.

Whey is a useful article in many cases. It is a pleasant and stimulating drink, with a certain food value from the lactalbumin, lactose, and mineral matter present. In some cases whey plus cream make an admirable combination.

The pure caseinogen of milk, now prepared by the Protean Company, in the form of a flour, and made into biscuits and bread, is an appropriate form of proteid administration. The taste, however, is an acquired one.

CHEESE.—There is no reason why cheese should be forbidden. The ill effects frequently attributed to it arise from the manner in which it is taken at the end of a meal, already excessive and badly assorted. Being a rich albuminous food, and varying in the proportion of fat present according to the variety of cheese, it should not be taken in large quantity; it should be well masticated, and it should be carefully distributed through the various vegetables or bread-stuffs of the meal (*vide* p. 248).

The composition of cheese depends entirely on the amount of fat it contains. There are three leading varieties—soft, hard, and skim-milk. A fair average for the fat and nitrogen present in these three types, *when fresh*, is as follows (Fleischman):—

	Soft.	Hard.	Skim-milk.
Fat	31 to 44	29	2 to 3
Nitrogen	13 to 24	28	19 to 33

The fatty acids present in cheese have been considered detrimental, but, on the other hand, it is possible that in some cheeses, especially the richer varieties, the acids formed by bacteria in them may be inimical to other putrefactive processes going on in the alimentary canal. If so, this would explain the absence of injurious effects from the taking of these cheeses noted in some cases of otherwise very weak digestion.

The soft cheeses include cream cheese, Wiltshire, Neufchatel, Gorgonzolas, and numerous other Italian and French cheeses. The hard cheeses, which ripen slowly, and are adapted for keeping, include the English and American Cheddar, Stilton, Cheshire, Gloucester, Dunlop, Parmesan, Gruyère, and most English, Dutch, and American cheeses. Gouda and Raden may be taken as illustrative of the skim-milk variety.

It is well to recommend patients who are very fond of cheese to partake of one of the softer varieties, as, although less digestible, they are much less likely to be taken to excess.

EGGS.—Eggs are an excellent dish for the gouty, and should form one of the staple breakfast dishes. They also constitute a very appropriate food constituent for the children of gouty parents, in whom the consumption of meat, and especially red meats, should be very limited indeed. It is well to remember that a hard-boiled egg takes three hours, and a soft boiled or raw one, from 1½ to 2 hours for complete digestion.

CARBOHYDRATES.—The fact that gout is unknown in countries like Japan, where a strict vegetarian diet is common, clearly proves that, as a class, carbohydrates can by no means be the direct cause of the disturbance of metabolism characteristic of gout. On the other hand, taken in considerable amount and with strong nitrogenous food-stuffs, their use is frequently accompanied by some evidence of local disturbance in the alimentary canal, or general disturbance of metabolism in the tissues, either of which may be characteristic of a gouty proclivity of tissue. When this tendency is pronounced, the sum of the local and general effects produces typical gout in a more or less acute form, the manifestations depending on the age and constitution of the individual. In other cases we find irregular gout, which, when well marked, may be regarded as equally typical, as it is dependent on internal peculiarity of tissue.

Carbohydrates, and especially those of the saccharine group, are as a class to be regarded as more potent noxious agents than meat. The explanation of this is probably found in not one, but many factors, which have already been alluded to under Etiology. A good rule with regard to them is to reduce the amount and simplify their form.

Saccharine foods and dietetic accessories, *e.g.* jams, marmalade, sugar, sweet cakes, are only to be partaken of occasionally and in small quantity, and in not a few cases, especially of stout adults, are to be studiously avoided.

With regard to the strict vegetarian diet so eloquently advocated by Haig and others, the good effects undoubtedly derived in many instances depend, in my opinion, on the simplicity of the whole diet, with the limited quantity of the chief nitrogenous ingredients, these being the two primary essentials in the dietetic treatment of confirmed gout. The following illustration may be given (Haig):—

Breakfast.

1 pint of milk.
Bananas
Apples
Pears
Plums, fresh, dried or cooked
Any other fresh fruit
ad libitum.

Dinner.

Much as lunch.
1 pint of milk.
1 oz. cheese.

Lunch.

Vegetable soup made with milk.
Plate of potatoes (with butter, oil, or milk).
2 oz. cheese, eaten with potatoes and any other vegetables in season.
Stewed fruit or tart.
Fresh fruit.
1 pint of milk drunk during the meal.

A close analysis of this diet, which is recommended for a person in health, shows that it is not quite so simple as at first sight apparent, and while a diet for the gouty framed on very similar lines is undoubtedly a very beneficial method of treatment in some cases, in others it is altogether unsuitable.

Popular belief, partly supported by medical opinion, condemns potatoes, but if used in moderation, and cooked and served with due precaution, there is no reason for prohibiting them, except in those special cases where they are definitely determined to be unsuited to the digestive capacity.

When new and moist they are indigestible; the best form is a well-boiled mealy potato in its skin, or the same put through a potato masher. A thoroughly well-baked potato is also good. When fried, or roasted in mutton dripping, or mashed with milk and butter, they are unsuitable in most cases. The other roots—turnips, carrots, parsnips, radishes, beetroots (also rich in sugar), artichokes, also cabbages, curly greens, Brussels sprouts, broccoli, and the green of cauliflower—should only be taken in small amount on account of their tendency to induce flatulence, etc. The following are more suitable: Spinach, flower of cauliflower, savoys, endive, lettuce, watercress, kale, leeks, onions, celery, cucumber, vegetable marrow, green peas, French beans. Asparagus has been condemned by some writers on account of the nucleins in the young shoots; also tomatoes and sorrel on account of the acids present; but, as many typically gouty subjects can partake of them freely, it is probable that these objections have only theoretical importance.

The green vegetables above mentioned can be freely partaken of in the form of salads, provided oily dressings and hard boiled eggs are avoided.

Mushrooms and truffles and other fungi are quite permissible in small quantities.

The pulses (lentils, peas, beans, haricot beans) are not as a rule advisable, because it is not an easy matter to make the patient realise that their nutritive value is such that their use must influence markedly the amount and quality of the other articles consumed. With regard to *puddings*, the simpler the better. Milk puddings, such as rice, sago, semolina, ground rice, etc., should be made without eggs in many cases. Suet puddings of all kinds are as a class to be avoided, but if made with bread-crumbs in place of flour, well boiled and unaccompanied by a heavy sauce, they may be taken without prejudice. If custards and omelettes, sweet or savoury, are taken, the nutritive value of the eggs must be recognised. Jellies, blanc-manges, lemon sponge, and creams may be taken in very sparing amount, and, as with other foods, a wise discretion is necessary both by the physician and by the patient. Fruits of all kinds in themselves are permissible, but must be taken with caution, especially in later adult life, and it is well to bear in mind the old saying, "Fruit is golden in the morning, silver at midday, and lead at night." Much depends on the amount of sugar used in the cooking and the accessories used at table.

BEVERAGES

To the confirmed gouty subject the question of what he may drink is sometimes a more important one than that of what he may eat. Alongside of this question there is another bearing on the time—relationship to meals—at which various liquids should or should not be taken. Thus, it is as a general rule advisable to recommend alcoholic stimulants only to be taken with meals; in other cases the consumption of fluids, of a non-alcoholic nature, may be wisely restricted mainly to the intervals between meals. Fluids may act prejudicially in two ways. In the first place, they may act injuriously in a mechanical way, *i.e.* by clogging the food elements, and at the same time diluting the digestive fluids, favour abnormal decomposition of the proteids and also carbohydrates. And, secondly, they may, in virtue of a specific action, lead to faulty nitrogenous metabolism in the digestive tract, and secondarily, in the tissues.

Like the question of diet it is impossible to lay down definite rules applicable to the disease. Everything depends on the age of the patient, his constitution, his previous history as to consumption of fluids of different kinds, the nature of the symptoms and the reaction of the tissues to various fluids. Specially is this so with the use of alcoholic stimulants. While there is no doubt that the subjects of inherited gouty tendencies are better without any form of liquor, this is frequently not so in the case of the patients more or less habituated to the use of stimulants. In judging of the suitability of the various liquors we must have regard to the usual methods of preparation of the individual beverages, *e.g.* beer, claret, champagne, etc., and to their common defects as recognised by experts in the trade. While these are outside the scope of this work, I think it well to indicate that their appreciation helps to explain the diverse views often expressed regarding the various liquors.

The free consumption of *water* can be safely recommended to many, but not to all, gouty subjects. In the case of stout adult plethoric subjects it may be advisable to restrict its use to early morning and late evening indulgence. The water is, as a rule, best taken on an empty stomach. The use of potash and lithia waters and various table waters are elsewhere referred to. With regard to *tea*, *coffee*, and *cocoa*, when suitably prepared these beverages may be partaken in moderate amount, but idiosyncrasies in their use are very often encountered. To some, cocoa is specially injurious, to others the daily consumption of coffee is soon followed by digestive and other disturbances, and in these cases special restrictions are called for. The amount of sugar allowed should be small. All *sweet beverages* should be restricted or cut off, more especially if they be in addition aerated. With regard to *alcoholic beverages*, points already referred to are of supreme importance. The decision as to what any given case can take, may only be arrived at after a careful study of the history, diet, and state of muscular activity in each case. There is no doubt that malt liquors and sweet wines are much more injurious than other liquors.

The sweet wines include champagne, Maderia, port, sherry, Malmsey, and Tokay; also porter, ale, and cider. Burgundy, Bordeaux, Rhine, and Moselle are almost void of sugar, and are therefore more suitable. The greater acidity of Burgundy and the Rhenish wines makes them, on the whole, less suitable than claret and Moselle wine.

German beer, *e.g.* lager, can frequently be taken with impunity when even one glass of the British beers will induce some acute disturbance.

MEDICINAL TREATMENT AND MINERAL WATERS

Some idea of the relative importance of medicinal remedies will have been gained from what has been already said in the general introduction to "Therapeutics." What we know with certainty may be summed up in the statement that all drugs which exert a normal diuretic action have a beneficial influence, and the greater the diuresis the more the benefit. Opinions will probably always vary as to the respective values of salts of potassium, lithium, and sodium, piperazin, urotropin, colchicum, salicylate of soda, guiacum, and other remedies, and individual experience will lead to perfectly honest but very divergent opinions regarding them. The more powerful diuretic action of citrate of lithium and citrate and acetate of potassium makes them occupy a relatively higher position than the various soda salts. Luff has recently expressed the view, based on experimental observations, that the salts of sodium are to be deprecated owing to an influence they exert in accelerating the transformations of the gelatinous biurate to the crystalline form. Sir William Roberts held a similar opinion. General clinical experience does not corroborate this view, *e.g.* it is not reconcilable with the benefit undoubtedly derived from the copious internal use of the Wiesbaden "Kochbrünnen," which may be regarded as a strong solution of sodium chloride. Nor is it supported by facts observed nearer home by many who use the sodium salts freely in combination with potassium or other salts, and it is perhaps wise to refer the greater value of potassium salts to their more powerful diuretic influences. An occasional short course of mineral waters is also an admirable measure. With regard to salicylate of soda my experience is in harmony with those who have not found the drug of any service in cases of typical gout. Of its value in some cases of chronic rheumatism, either when taken alone or in combination with quinine or other tonic, there can be no reasonable doubt, and it is advisable to give it a trial in doubtful cases. A useful mixture in some cases is Sodii salicylas, lithii salicylas, potassii citrat. āā gr v. t. id. Various made-up combinations with colchicum lately in the market may also be tried, but much need not be expected from them.

Salol (5 to 10 gr. t. id. on an empty stomach) and salicylate of bismuth (20 to 40 gr.) may also be useful in cases with subacute gastro-intestinal disturbance, but these must only be looked upon as adjuncts to appropriate treatment, and their use can only be necessary as the result of faulty dietary, usually combined with imperfect evacuation of the bowels. Guiacum has been well spoken of, but its taste seriously interferes with its recommendation. The special value that attaches to the use of sulphurated waters leads to the belief that the continual administration of small doses of sulphur might be attended with marked benefit; and I have lately seen a patient, a shrewd man of affairs, with pronounced uratic deposits in the palms of both hands, and an unusually extensive knowledge of health resorts both in this country and abroad, who assured me that he had of late derived great benefit from the use of this remedy in the real old-fashioned way—a bag of sulphur in his trousers pocket. He assured me that marked relief of the pains had followed this treatment, and that no benefit had accrued until his scarf-pin showed marked discoloration. Such stories have to be received with caution; but we know so little about the rôle of sulphur in the animal economy, that no harm will accrue from our keeping our minds open to new suggestions, especially if these prove to have any basis in clinical experience.[1] In the meantime we can sum up by emphasising the fact that the medicinal treatment of gout is by far the least important part of treatment and at the same time we must indicate two dangers attendant on their use. In the first place, undue stress is often laid by the patient, more especially in the earlier stages of the disease, on the drugs in use, to the detriment of the more important dietetic regime necessary; and, in the second place, the excessive use of alkalies, mineral waters, salicylate of soda, etc., occasionally does much harm by the general depression attendant on their abuse.

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[1] It is quite possible that this old-fashioned and apparently unscientific treatment may not be so unscientific as is imagined, *e.g.* compare the most recent method of treatment of syphilis based on the fact that the mercury is absorbed by volatilisation, and not at all by inunction.

In the selection of a general tonic for gouty subjects, iron should as a rule be avoided, as it very often disagrees.

MINERAL WATERS

The various waters in common use are recommended for one of the following reasons:—

i. A purely purgative action.
ii. A diuretic action.
iii. A diuretic and medicinal action.

Of the Purgative Waters.—Franz Joseph is one of the most palatable, and its action is mild. Apenta, a bitter and faintly sulphurated water, is also mild and much favoured. Hunyadi Janos, Æsculap, Friedrichshall, and Carlsbad are all bitter aperients, with a more powerful action, but are all suitable for occasional use. The action of these waters is hastened by admixture with hot water, and they should be taken the first thing in the morning. The continual daily use of any mineral water or saline purgative is not, however, desirable.

Of the Diuretic Waters.—Some are palatable, and are taken as table waters; others are specially suited for a short course of home treatment, and should be taken thrice daily on an empty stomach for a period of two or three weeks. The table waters, which are aerated, include Apollinaris, Briresborn, Johannis, Seltzer, St. Galmier, and Salutaris, which is a distilled water of English manufacture. The other group is represented by Contrexéville, a mild water containing sulphate of lime and magnesia, and Vichy water (bicarbonate of soda the main saline), of which there are several varieties. Célestins is the best known, but the Grand-Grille and Hauterive may also be employed with benefit. Sulis water from the springs at Bath is also useful.

Diuretic and Medicinal Action.—Occasionally the presence of anæmia, rheumatism, or some pelvic disorder suggests the use of other waters. Thus Levico (arsenic and iron) is useful in anæmia. So also is Schwalbach (Weissenbrunn preferred) and La Bourbule, the former being especially useful in rheumatic cases. Kissingen, a saline gaseous aperient water, is valuable in various uterine and other pelvic disorders in gouty subjects.

The foregoing only represents a few of the mineral waters available; artificial representations of many of these and other waters are prepared in this country and sold in tabloid form.

SPA TREATMENT

The beneficial effects undoubtedly derived from spa treatment in many instances arise from a variety of circumstances. For the time being worries and cares are lost sight of, the mind is at rest, and more or less pervaded by that feeling of hopeful expectancy that is of such paramount importance in treatment. The change of air and surroundings, the greater attention to diet, and the general feeling of *bien être* engendered by the interests and amusements which form an integral part of every well-regulated spa, all tend to favour that return to health which has as its most striking manifestation a re-establishment of a nitrogenous equilibrium. The great importance of the skin as a channel for the elimination of urea and other waste products has already been referred to. The local measures associated with a course of balneological treatment may best be indicated by illustrative cases.

The *choice of a spa* will depend on various things, the most important being the temperament of the individual and the special manifestations of the disease which the patient exhibits. It is needless to recommend patients to whom excitement and amusements are part of the breath of life to go to one of the quieter health resorts, while on the other hand there are not a few to whom a small quiet place is a much greater attraction. A very short summary can only be given here.

For Articular Gout.—The thermal waters of Bath and Buxton rightly maintain a high position, Bath being more suitable for the spring and autumn months on account of its mild climate, and Buxton more appropriate in summer owing to its more bracing air. Woodhall Spa (Lincolnshire), with its bromo-iodine waters, and the saline sulphurous chalybeate waters of Llandrindod (Radnorshire) can also be highly commended. The brine baths of Droitwich (Worcestershire) are of peculiar value in reducing the stiffenings and deformity arising from recurrent articular attacks. The Droitwich waters are only available for external use. Aix-les-Bains, Aix-la-Chapelle, and Schlangenbad occupy a deservedly prominent place in the list of foreign spas.

Chronic and Irregular Gout.—In addition to those already mentioned, we have in this country Cheltenham, Lamington, Harrogate, and Strathpeffer. The sulphurous waters of Harrogate, Strathpeffer, and Llandrindod, are especially useful in gouty affections of the skin and mucous membranes. Lamington is well spoken of for cases of gouty glycosuria. The various foreign spas in virtue of their varying chemical composition lend themselves more readily to further differentiation. Thus we find Vichy, Homburg, Kissingen, Vals, and Wiesbaden especially useful in cases of chronic gastric and intestinal derangements. In states of plethora with hepatic torpidity, Carlsbad and Marienbad are more appropriate, and for urinary disorders Contrexéville, Neuenahr, and Vichy, deservedly occupy a high place. The sulphur waters of Aix-la-Chapelle, Aix-les-Bains, Baden (Switzerland), and Soden (foot of Taunus mountains), are good for cutaneous disorders and catarrhal states of the various mucous membranes. Soden is a quiet, little, attractive place practically unknown to English visitors, but its numerous springs compare most favourably with those of much better-known places. Schwalbach and Pyrmont are two other very attractive health resorts, and are especially good for anæmic, debilitated, gouty subjects. In view of the great value of medical gymnastics, especially when combined with balneological treatment, it may be well to mention the leading spas which have a fully equipped mechanico-therapeutic institute. These are Aix-la-Chapelle, Baden-Baden, Wiesbaden, Wildbad. It is unfortunate that in this country, where gout has, as it were, its headquarters, more attention is not paid in our medical schools to instruction in dietetics and in mechanico-therapy—the two fundamentally important factors in treatment.

Acute Gout—Recent Attacks

These cases are the most suitable for the attainment of an excellent result, and the more recent the attacks the more hope there is of a speedy and permanent beneficial result. Needless to say the details of treatment will vary, depending on the constitution of the patient and the nature and strength of the waters employed. The course should last not less than five or six weeks, and its daily routine should be along the following lines :—*Diet.*—The dietetic treatment should be conducted along lines already laid down, the food being simple, and composed mainly of light animal foods, eggs, vegetables, and the more easily digested bread stuffs. Preferably no alcohol or wines should be taken, but if that is not feasible, whisky and potash, or lithia water, or a little sound claret are the most suitable. On no account should liquors be mixed. *Exercise.*—Ample exercise should be taken, a course of passive movements supplementing the patient's own exertion in the matter. There is much more danger in delay in moving the affected joint than there is in unduly exercising it at an early stage. In the case of the upper extremity there is greater necessity for a special course of passive movements. *Baths.*—If patient is a robust subject, he should take a curative bath daily, but in weakly or nervous subjects every other day will suffice, at any rate at the outset. A feeling of slight excitement, a sense of general discomfort, loss of appetite, or sleeplessness, are indications pointing to the advisability of prolonging the intermissions, or it may be, diminishing the temperature and strength of the bath. During this treatment it is not an uncommon experience for acute pains to develop in the affected joints, even a further acute attack may supervene. Such occurrences are usually ascribed to the searching nature of the remedy, and if not immoderate need not be regarded with disfavour. The best time for the bath is the early morning, an hour to an hour and a half before breakfast, and it should be taken on an empty stomach. (To those patients who cannot or will not bathe fasting a cup of coffee or tea may be allowed.) The temperature of bath ranges from 80° to 105° F., and depends on the susceptibilities of the patient. He should remain up to the neck in water, and continue to exercise the affected joints by active movements, and also by friction and kneading of the parts. The duration of the bath should be from eight to ten minutes to start with, and be gradually increased up to twenty or thirty minutes. The duration, however, largely depends on the temperature of the bath. After drying and using moderate friction with a roughish towel patient should lie down for half or three-quarters of an hour, and thereafter dress himself in clothing appropriate to the time of year. He will then be ready for his breakfast. The warmer baths should only be used for stronger and more vigorous subjects, as they are liable to upset more weakly or nervous ones. *Drinking Cure.*—This consists in drinking the waters in an amount varying from one and two pints thrice daily on an empty stomach, *e.g.* 8 A.M., between 11.30 and 12.30, and between 4 and 5 P.M. As a rule no further measures are called for, but the use of the douches and various forms of local hot-air or other applications are useful in appropriate cases.

A Case of Chronic Gout with Irregular Manifestations

Here the regulations already laid down for diet and exercise should be even more stringently enforced. The latter measure especially is only too frequently not given adequate effect to in spa treatment. Carbohydrates and sweets must be reduced, or even abolished, and great care taken to avoid admixture of fruits and carbohydrates with the chief proteid foods. A large quantity of water should be drunk in the course of the cure, beginning, however, with small amounts (½ to ¾ pint thrice daily), and every third or fourth day gradually increasing up to two or even more pints thrice daily. The amount should be carefully regulated, and is largely dependent on the cardio-vascular tone of the individual. Special care should be taken with stout subjects, who are also less liable to take the amount of exercise requisite daily. The baths should only be taken every alternate day, or in some cases less frequently, but in the intervals various accessory local measures are of the greatest service. These comprise the douche (*vide* vol. i. p. 436), active and passive movements of various kinds, which are most readily obtained by a course of exercises in a well-equipped Swedish mechanical institute, and various forms of local hot-air treatment. A very good general rule to adopt with hydropathic and allied remedies intended to influence local metabolism is to use them "little and often."

Hydro-therapeutic measures are not as a rule applicable in cases of gouty cachexia, nor in elderly persons especially with obese tendencies, and further, their use requires special consideration in cases where any organic, cardiac, renal, or pulmonary lesion exists. The degree of general limpness and enervation so frequently experienced in the earlier part of the course reflects the profound alterations that are taking place in general metabolism, and these may be still further indicated by the occurrence of a marked oxaluria or phosphaturia. After a course of spa treatment it is often desirable to endeavour to thoroughly establish the cure by sending the patient to some alpine or other health resort for a time, where the climate is thoroughly invigorating and calculated to promote general tone and vigour to the system.

TREATMENT OF ACUTE GOUT

Acute gout falls to be treated on the general principles applicable to other inflammatory states. The indications are to relieve the constitution, and to moderate the local inflammation, both of which are best fulfilled by constitutional rather than local treatment. Rest in bed, free purgation, and a low diet, are the essential constitutional remedies, the two latter being employed in proportion to the acuteness of the symptoms and the constitutional vitality of the individual. In weakly and in old subjects, and in cases where the gouty cachexia is pronounced, the treatment must be less depressant, and a more generous diet with some alcoholic stimulant may be indicated. On the first suggestion of the paroxysm in an otherwise healthy subject, a full dose of calomel (4 to 8 grs.) and colocynth (2 to 4 grs.) should be taken and followed by a saline cathartic (Carlsbad salts). In a few cases this treatment suffices to arrest a paroxysm, but if the attack is fully developed its remedial influence is not so noteworthy. Recourse should then be had to active diuretic treatment and the administration of colchicum. Large doses of lithia or potash water should be taken at least three times a day, and in addition fifteen to thirty minims of the vinum or tincture colchici should be given in a little water, or in the following mixture thrice daily:—Vin. colchici ℥ss., pot. citrat. ℥ss., lithii salicylas ʒj., aqua ℥vj.; ℥ss. in water thrice daily. Great care must always be exercised in the use of colchicum, as not a few subjects react strongly to its influence, severe general depression or diarrhœa resulting from even moderate doses. How the drug acts is unknown. There are no conclusive indications that it influences the excretion of uric acid. The very slight increase noted by His in the most recent observations on the subject are not (*Deutsch. Archiv für klinische Medicin*, Oct. 1899) distinctive, and we may confidently assert that the beneficial effects of the drug are not dependent on any direct influence on uric acid. Sir Dyce Duckworth has suggested that the result may be explained by the cholagogue action of the remedy. Salicylate of soda is another remedy that has been found useful in a few instances of acute gout when colchicum was unavailing. In the few cases that I have tried it in acute gout I have never been satisfied that it exerted any therapeutic influence. Ten to fifteen grs. of Dover's powder, or a combination of bromide of lithia (30 grs.) and chloral hydat. (30 grs.) may be indicated for their sedative and hypnotic action. This treatment must be carefully revised from day to day in the light of the patient's general state, and notably the condition of the pulse, which may be taken as a reliable guide as to whether the depletent remedies are being administered in excessive amount. The diet should be limited to milk only for the first few days, or milk alternately with weak beef tea. Solid food of any kind is better withheld in robust subjects—nothing but benefit will accrue from a mild starvation diet. In a few days bland farinaceous food may be added, provided the state of the tongue is satisfactory, and all flatulence and eructations gone. Gradually, white fish, boiled chicken, eggs, and easily digested bread stuffs, are added to the diet. With regard to local treatment, elevation of the limb, a covering of wool surrounded by oil silk, a bandage, and a cradle to protect the limb from the weight of bed-clothes, are usually all that is required. Of other measures the most appropriate are flannel fomentations wrung out of hot water and sprinkled freely with laudanum, or the application of lint soaked in lin. belladonna and tinct. opii. When these are not available, lint soaked in methylated spirit or one of the cheaper whiskies makes a fair substitute. All cold applications, blisters, leeches, and the like should be avoided. Dr. G. Balfour has found the application of steady manual pressure to the joint from the outset of symptoms to be attended with good results. Such drastic measures will probably, for obvious reasons, have only a very limited application. As soon as all acute manifestations are gone, active and passive movements of the joint should be begun steadily and persevered in. In subacute attacks the various troublesome local complications met with, *e.g.* thrombosis, œdema, eczema, etc., must be treated on general medical principles, but their too zealous treatment is to be deprecated.

ILLUSTRATIONS OF TREATMENT

From the foregoing outline of treatment it will be evident that the therapeutic measures vary enormously with the different stages and very diverse manifestations of the disease. As previously stated, every case of gout is a new problem in therapeutics. I have thought it well to give further point to this by recording in detail a series of illustrative cases, and have selected a few which have been under my observation for a lengthened interval, so that I am in the position to speak with accuracy of the effects of the different regime recommended in each case.

1. Mr. A., 16, schoolboy.—Marked hereditary history of gout.
    i. Erythematous eruptions.
    ii. Erythema elevatum diutinum.
    iii. Pharyngitis and tonsillitis.
    iv. Nail disorders.

This case is selected to illustrate some clinical features of gout in the young subject and also to emphasise the rational prophylactic treatment of the disease. The patient is a tall, big-boned, muscular subject whose weight is distinctly above the average for his height and years. He excels in athletics, being an adept football player, golfer, and swimmer. His home surroundings have been of the kind most favourable to the full indulgence of a keen appetite and sound digestion. His diet is plentiful and rich, and all his life he has been in the habit of drinking large quantities of milk independently of much nitrogenous food at ordinary meal times. Scotch oat cakes, butter and jam are a special weakness. He is a total abstainer.

About the age of 8 or 10 he began to show a tendency to the frequent development of "skin spots," whose onset seemed to coincide with a slight excess in diet. From the descriptions available there seems to have been discrete erythematous patches, but, whatever their nature, they soon disappeared under the influence of a purgative and restricted diet. . Three years ago he was the subject of a pronounced attack of erythema, affecting especially the fingers, back of wrist, and to a less extent the knees and upper part of face. These parts were swollen, red, burning hot, and extremely itchy. The patches on the back of the wrist were markedly raised and corresponded closely to the conditions described by Crocker, "erythema elevatum diutinum," as occurring in gouty subjects about the age of puberty. The conditions disappeared under the simple treatment above mentioned. Since that time patient has had no similar attack, probably because, on the slightest appearance of it, which is by no means infrequent, the necessary therapeutic measures are taken. He has found by experience that if a saline be taken sufficiently early the other remedial measure is not necessary, and he acts accordingly. The reliability of his own observations and the extent of his faith may be gauged from the fact that he seldom goes off for a holiday without a supply of his favourite mineral water. On more than one occasion when he has done so, he has felt seriously out of sorts within a very few days, the most striking *objective* symptoms being the development of swelling, redness, and pain in one or more fingers. I have not seen this condition, but his mother, who has an accurate lay knowledge of the disease, affirms that it is "either gout or chilblains." She recognises no relationship between the two, and, when her inclinations lead her to write for a remedy for chilblains, all her knowledge of facts prompts her to adopt the remedies appropriate to gout. The patient is also subject to occasional attacks of inflammation of the pharynx and tonsils, the inflammation lasting for 24 or 48 hours, and resembling more the appearance of a general swelling and acute congestion than a bacterial invasion. A year ago he had some trouble with his nails. A transverse furrow developed at the roots of two of his toe nails and one finger nail, and gradually the nails were cast off and replaced as after a traumatism.

With regard to the diagnosis of gout in this case, while some may question its accuracy, I am perfectly satisfied that the manifestations of disease described are directly due to the patient's gouty proclivities. It is a case of gout in the young and robust subject.

The case admirably illustrates the treatment appropriate to the disease. It is clear that his symptoms tend to develop after a slight or marked excess in diet, more especially if jams or other sweets have been largely consumed, on which occasions there have been no abatement in the amount of red meats and other nourishing foods. The saline, which may sometimes be associated advantageously with blue pill, acts beneficially in two or three ways. It removes from the alimentary canal certain toxic substances produced locally as a result of defective metabolism, which act directly as a local irritant, and by their removal as well as by the withdrawal of any other undigested foodstuffs in the alimentary canal a condition of rest is established in the general cell metabolism, which enables the tissues to cope more successfully with the surfeit of nutritious matter to which they have been subjected. This latter is rendered more easy, if, as is generally the case, the patient's diet for the few following days is a more strictly physiological one. The depletion effected by the rapid withdrawal of some fluid by the alimentary canal doubtless assists the re-establishment of a general equilibrium. The occasions of these various congestive attacks in a young subject are probably, as with the paroxysms in adults, to be explained as the result of long-standing over-nutrition, with deficient elimination, and the excess immediately preceding the attack has merely strained the capacity of the tissues—intestinal viscus and general cell life—to breaking point. The general plan of treatment appropriate to such a case may be briefly summarised—

i. Moderation in diet—a slight all round diminution of the total quantity of food consumed at each meal; the acquirement of self-control.

ii. Restriction and gradual cessation of the milk drinking between foods, and also a diminution in the amount of fluid drunk at meal times. His habit is to drink very large quantities of fluid at the different meals, which fluid would be more wisely taken in the intervals.

iii. Limitation of the jams, sweets, and fruits, of which he partakes inordinately, also care in the consumption of the various sweet summer beverages. (These patients are as a rule addicted to sweets of different kinds.)

iv. Daily satisfactory evacuation of the bowels, by artificial means if necessary. In any case, to be supplemented by an occasional saline (once in 8 or 10 days).

No other restrictions are called for. Nor are any recommendations as to exercise, hydropathic or other measures, the least necessary so long as the patient's inner respiratory activity is at the high level entailed by his being in first rate physical training. However, it is well again to refer to the fact that the patient finds that strict attention to the last of these directions alone suffices to keep him free from any active manifestations of his gouty tendency, and he acts accordingly. This circumstance in no way invalidates the importance of the other recommendations, the neglect of which will certainly, in course of time, with the altered habits incidental to adult life, lead to other characteristic developments of a minor or major type. Succeeding years of indiscretion progressively increase the hold of the disease on the tissues, and further experience of the case may illustrate that the efforts of nature, even when aided by a much more strict regime than that just recommended, are quite incapable of extinguishing the disease.

2. Mr. B., æt. 26, mason.

Chronic articular gout.
Lithæmia.
Acute exacerbations.

This case is selected to illustrate some points in the dietetic treatment of the disease in its more acute phases. The patient was a hospital one, and a full record of his case with a series of laboratory observations made on his general metabolism are in course of publication (*Journal of Pathology*, summer 1900). I have not the same knowledge of the later history in this case as of the others; but the points that I wish to emphasise are sufficiently brought out by the notes available. Unlike the others recorded, this patient was in the habit of passing large quantities of free uric acid, and he was thin, weak, and slightly cachectic in appearance. The salient features are as follows. The extent of his weakness may be gauged from the fact that two months after the acute attack the record of the dynamometer gave—R. hand, 55; L. hand, 35:—

The hereditary history showed the father to have been subject to rheumatic fever, the grandfather to have been rheumatic, and one brother to be subject to "attacks like the patient." He enjoyed good health until fourteen years of age, when he had a "rheumatic" attack lasting three weeks. When æt. twenty, similar although less severe attacks developed at intervals, and when æt. twenty-three a specially severe one lasted thirteen weeks. When æt. twenty-six, patient was admitted to hospital suffering from well-marked acute polyarticular manifestations, supervening in joints and tissues already the seat of chronic deforming gout. The ankles, knees, hands, elbows, and ears were all involved, crystals of urate of soda being readily obtained from the deposits in the ears. Patient had been a fairly heavy meat eater, a temperate drinker, addicted to very little exercise, and accustomed to the exposure necessitated by his trade, which entailed a considerable amount of travelling from place to place. He did not think that diet had played an important part in the manifestation of symptoms, but he attributed an influence to the drinking water of various localities. Tomatoes were a special weakness, and he had partaken freely without apparent detriment.

After recovery from the acute attack patient was gradually put on a light hospital diet, as follows:—

Breakfast.—Porridge and milk, toast, tea, and frequently an egg.
Dinner.—Soup, bread, and fowl, fish, or red meat, with potatoes; or fish, fowl, or meat and potatoes, with a pudding; occasionally green vegetables.
Tea.—Tea, bread and butter.

And in addition a cupful of milk was taken either alone or with soda water twice daily. During this time patient was making a very slow recovery, being continuously very subject to sharp recurring pains in different regions, excessive weakness in the feet, hands, etc., and he was in this state when he left the hospital to go to the Convalescent Home for four weeks.

His diet then was as follows:—

1. Porridge, tea, bread and butter.
2. Fish or fowl, bread, potatoes, rice or other milk pudding.
3. Tea, bread and butter.
4. Porridge and milk.

The most important differences between this and his former diet were the absence of all soups, red meats, and green vegetables. Under this regime he improved very materially, and, for him, very rapidly. The pains diminished and soon disappeared; he gained 6½ lbs. in weight in four weeks. He now felt well.

On his return to hospital for further observation his diet was inadvertently changed back to his former hospital diet with immediate unfortunate results. The pains reappeared, the stiffness and weakness became accentuated, and at least one of the former weak spots became swollen, red, and tender within thirty-six hours after his return to hospital. His appetite and digestion remained apparently unaffected, the tongue was slightly furred, and the total amount of food consumed was actually less in amount than it had been at the Convalescent Home. No actual acute attack developed, but his whole metabolism was deranged and he lost 3¼ lbs. within six days. Even allowing for the change of air incurred on his return to the hospital, the case was a perfect picture of the profound influence of diet on the disease. If we analyse the altered circumstances which led to the derangement of metabolism, there is no doubt that the soup was prejudicial, and mainly because it interfered with the normal local metabolism in the proteids in the diet. There is also every reason to believe that the milk taken in the course of the hospital diet was not calculated to give the various glandular secretions the rest requisite for their due functional activity. Other points there may be, but these will suffice. In gout, as in health, the tissues can cope with a range of diet within which no untoward effects result. The "convalescent" diet was the more physiological one, and a reference to its nature and amount clearly shows what nature is willing and able to do. Further, that diet could certainly be modified in different directions, *e.g.* to include vegetables without detriment so long as the necessary rearrangement was made.

There are not a few cases like this one in respect of the noxious influence of soup, but in this connection regard should be paid to the points in the composition of soup already referred to. With regard to the meat, this patient is an illustration of a type in which all red meats are better avoided. Whatever the exact cause may be, there are cases of gout when the reaction of the tissues to intestinal influences is more marked and more unfavourable if red meats are a component of the diet, and these should be dieted accordingly. But such cases are exceptional.

3. Mrs. C., æt. 32, married; 2 children.—Hereditary history of gout.
    - i. Neuralgia.
    - ii. Pains in great toe joints and legs.
    - iii. Acute gout.

This patient is a big, well-built woman of active bodily and mental habits, with keen appetite and excellent digestion, which she is in a position to gratify, although she does not eat immoderately. Her weight well exceeds the average for her height and years. She occasionally has a glass of claret to dinner, and takes very exceptionally a glass of beer to lunch. The latter is a beverage of which she is particularly fond, but she indulges very occasionally, as she has long recognised that it was prejudicial to her. This case illustrates well the ill-declared type of gout that is more frequently seen in Scotland than the truly typical form.

As a girl she was subject to facial neuralgia, which, however, has quite disappeared. Since the age of 17 she has been occasionally the subject of sharp pains in the calves of the legs, and eighteen months ago she was confined to bed for two days with severe pains in the upper arm and shoulder, a condition which I was satisfied was a slight inflammatory attack involving the brachial plexus. For many years she has had occasional sharp twinges of pain in both great toe joints, lasting from five minutes to half an hour, often associated with a stinging pain in the calf of the leg and cramp-like feelings in the soles of the feet. She has also been a martyr to chilblains. Six months ago a typical mild attack of acute gout developed in the left great toe joint, which was recovered from in less than three days. The attack developed at 10 P.M. and only lasted some five or six hours, and when I saw her at the height of the pain the affected toe joint was slightly swollen, the superficial veins prominent, the skin showing a localised red area the size of a shilling, and the joint very painful on pressure. On this day she had partaken of a larger lunch than usual, and with it a bottle of beer, and to this indiscretion she attributed the attack, although she has occasionally taken beer without any accession of symptoms. That beverage is a more active poison to her than to many others of apparently equal gouty proclivity.

The diagnosis is quite assured in this case, and the earlier manifestations of neuralgic pains, etc., may be compared with case 1, where the earlier symptoms were more vascular in origin. It has long been recognised that a proneness to inflammatory conditions of the peripheral nerves, and to a weakness in the vascular system (which may, of course, be partly of nervous origin) are characteristic of inherited gout. This relationship has been emphasised by Hutchinson. Care must, of course, be taken in these nervous cases to differentiate rheumatism, and while this may be difficult and often impossible, in not a few cases an honest and accurate diagnosis of gout may be arrived at.

What is the appropriate treatment? We have here an adult woman in comfortable circumstances, living a town life, with a husband and two children to look after, accustomed to good living, and not in the habit of always taking exercise proportionate to the full diet enjoyed.

In view of these special circumstances we must not be surprised if good results are less readily obtained, nor will it be surprising if more attention requires to be devoted to promoting the functions of the excretory organs and getting rid of the accumulated products of deficient combustion in the body.

These can be judiciously influenced by exercise as illustrated by the following :—Some years ago a friend of the writer, a typical example of plethoric gout, sought advice from her consulting physician for various acute gouty manifestations to which she was a victim. She was recommended a very strict diet, and one which could not easily be given effect to even in a large, well-regulated household; she declined the recommendations, stating that she would rather die, a statement which those who knew her appetite and mode of living could quite believe. She then discussed the matter with an intimate lay friend, who suggested that she should not over-eat, and should buy a bicycle and ride it. She accepted the recommendations, regarding the former as in the light of a compromise with the advice of her medical adviser, and from that time until now she has been remarkably, although not entirely free from all active manifestations.

The special treatment may then be summarised as follows—

(i.) A slight all round restriction in the quantity of food consumed. The lunch to be mainly if not always a vegetable one, and red meats to be replaced by chicken or fish for dinner, at least one day in the week. Sweets of all kinds only to be partaken of sparingly. The principal meal to be taken dry, one small glass of claret to be taken at dinner if desired. (This is the only wine for which she has a liking.) Beer is better avoided in this case. Water to be drunk freely between meals, and a tumbler of hot water taken at night.

(ii.) More systematic exercise. Sharp walking or cycling in place of driving, supplemented by ten minutes' physical exercise daily, preferably after morning bath.

(iii.) Attention to the bowels. As the bowels move daily without an aperient, all that is required is the use of a mild yet active saline every week or ten days. Further measures will probably be necessary at the monthly periods, as the patient, like many others of a like habit of body, is prone to undue states of depression in the premenstrual period.

(iv.) Hydropathic Measures. If the foregoing measures are duly carried out, the only hydropathic measures called for will be fulfilled in the daily morning bath followed by active friction, supplemented by a hot bath once a week. But as a matter of experience, the first three measures are only imperfectly carried out, with the result that more active eliminative treatment becomes a necessity. The choice of a spa is in the main immaterial so long as the important elements of cure are secured. These are, a complete change of air and surroundings in a climate where temperature and other atmospheric influences will conduce to a reasonable amount of muscular activity, a simple diet, judiciously flushing out the system by a quantity of one to three pints or more fluid daily at intervals on an empty stomach, and a course of baths two to three times a week, adapted to promote vigour and tone in the skin. Special care has to be exercised in the amount of water to be drunk, and the number and form of the bath to be recommended in stout flabby subjects. While this patient is not flabby, she reacts unfavourably to even a mild Turkish bath, and has also always reacted badly to cold sea bathing.

4. Mr. D.,[1] 38, tailor.—Chronic tophaceous gout; Surgical interference.

I have selected this case because it illustrates so well some other points in the management of gouty subjects. It is additionally interesting owing to its severity, such a case being almost unique in a Scottish hospital. My attention was first drawn to it by Mr. Alexis Thomson, under whose surgical care he was, and to him I am indebted for the record. During the past year I have taken an opportunity to see the patient frequently, and obtain from time to time an accurate record of the results of treatment. The successful results derived from the limited therapeutic measures employed clearly indicate that any line of treatment found beneficial in a given case of chronic gout should be considered applicable only to the individual in question, and no generalisations whatever should be made from it applicable to the disease. There is no single line of attack on the disease. Every case of gout has to be considered on its own merits.

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[1] The surgical aspects of this case are recorded in the *Edinburgh Hospital Reports*, vol. vi. "Gouty Formations in Tendon Sheaths, Bursae and Skin (with Photographs)." By Alexis Thomson.

The patient is a small, thin, rather pale, but not cachectic subject. There is no hereditary history of any importance, but the patient knows very little about his family. As a young man he was a keen football player, and partook freely of port wine and whisky. His diet has been the ordinary mixed diet of his class, the only noteworthy point being his strong aversion to fats. His occupation conduces to a very sedentary life. About the age of 28, shortly after the end of his football career, he had his first experience of acute gout, which involved the ankles. About eight or ten months later he had a second attack, involving the wrists and back of hands, and thereafter he had an attack about three times a year, each one incapacitating him for about two or three weeks. During these attacks he was kept on a light febrile diet, but in the intervals his food was an ordinary mixed diet, with occasional indulgences in liquor. Two years ago he was off work for seventeen out of the fifty-two weeks of the year, this period representing at least three severe attacks. At this time, even in the intervals, he was much troubled with pains and discomfort in connection with the numerous gouty deposits that were now developed, the position and size of the swellings interfering with the sitting position adopted by tailors, and also with his sewing powers. About eighteen months ago his medical adviser suggested surgical interference, and he was admitted to hospital for this purpose. He then presented all the typical features of a very pronounced case of chronic tophaceous gout. The ears were the seat of numerous characteristic gouty deposits. The extensor aspects of several fingers, especially those of the right hand, most used at his work, showed the presence of nodular swellings, varying in size from a small bean to a small marble. It was not easy to determine whether these were truly subcutaneous or connected with the tendon sheaths. The second joint of the right middle finger was enlarged, swollen, and tender. The subcutaneous tissue of the feet and ankles was the seat of several large swellings, varying in size from a marble to that of a small orange, one of which had burst and exuded crystals of urate of soda. A similar swelling was present over the left olecranon process. The joint structures as a whole were remarkably free. Five of these tumours were removed, two from the neighbourhood of each ankle, and one from the elbow. They consisted of masses of urate of soda crystals, embedded in thickened and degenerated connective tissue.

Patient has enjoyed very much better health since the operation, and he has not been a day off work for more than a year. His regular diet has not been materially changed, but both he and his wife agree that he is now almost a total abstainer. His diet is now as follows—

Breakfast.—Porridge and milk. One and a half breakfast cups of tea, with moderate amount of sugar. Bread and butter, and usually an egg or fish.

Dinner.—This may be either soup (broth, rice, or potato) and meat (boiled, stewed, or steak), or meat and a pudding (suet puddings of all kinds and rice puddings). Green vegetables are taken probably two days a week; potatoes are taken sparingly, and bread in fair amount.

Tea.—An egg or fish; bread, toast; one and a half breakfast cups of tea, and frequently jam.

Supper.—Glass of milk, often with a little bread and butter.

In addition to the foregoing he drinks large quantities of water between his meals, and under this regime he now enjoys practically perfect health, although the right middle finger may occasionally give him a little discomfort, otherwise the tophi originally present have remained unaltered. No hydrotherapeutic or other special remedial measures have been employed, for obvious reasons, and yet the results of treatment must be regarded as highly satisfactory. Such a result must, however, on no account be interpreted as minimising the great value of hydrotherapeutic and other measures calculated to stimulate the eliminative powers of nature and promote a more healthy tone and general metabolism. It is probably more wisely interpreted as indicating that when nature is relieved from the injurious effects of even one noxious agent (in this case the alcohol), she may find herself competent to restore a fair nitrogenous equilibrium on a rational mixed diet. In this case surgical measures certainly assisted to give him a fresh start in the path of normal metabolism.

5. Mrs. E., 55.

    i. Peripheral neuritis and vascular disturbances.
    ii. Lumbago.
    iii. Headaches.
    iv. Tophi.

Mrs. E. is a stout, typically gouty-looking subject, of plethoric habit, who has lived an active town life and has lived well. Of late years she has restricted her diet slightly, more, however, in accord with the teachings of her own experience, than as a result of medical advice which she seeks, but does not readily follow. There is a distinct hereditary history, a brother and two uncles being afflicted with well-defined gouty manifestations. Patient has on the whole been a very healthy woman; she has had ten children.

When thirty years old, and after the birth of her second child, she was the subject of periodic attacks of very severe pain in the sole of one foot. This pain would last from two to four hours, and completely incapacitated her. During the paroxysms the foot looked bloodless, and felt cold, and was usually treated by being placed in very hot water, followed by elevation of the foot above the rest of the body. The onset of these attacks could not be accounted for, but occasionally one developed after over-fatigue. Pains of a somewhat similar, although much less severe nature, are occasionally complained of now, but patient believes that these are prevented, or very much relieved, by the continuous wearing of a tight foot bandage, which she has worn for more than twenty years. The conditions resembled that now described as metatarsalgia (see vol. ii. p. 447). She has had several severe attacks of lumbago, which have usually occurred towards the end of the summer months, and have been associated in the mind of the patient as the penalty of indulgence in fruits of various kinds. This association has been present sufficiently often to attract her own attention, but it has not influenced her habits very much, and for the last five years she has quite expected the annual visitation, which, however, has been of a mild character. Her vaso-motor system has long been in an unstable condition. When fatigued, or more readily on dining, especially if the meal commence with a hot soup, a red flush (one or more), develops on the cheek, nose, or chin, and remains for some time. Of late years she has been subject to very severe headaches, and about a year ago, patient came under my observation owing to the development of a small tophus over the sixth rib on the right side, which had burst quietly, and was exuding crystals of urate of soda. An examination of urinary and cardio-vascular systems reveals no evidence of chronic kidney disease (sp. gr. 1016), but a careful consideration of her history and constitution led me not to lay much stress on the absence of external evidence of renal weakness. Her menstrual life had been characterised by frequent excesses, but never such as to call for special treatment, as she was fully aware of her plethoric habit of body. One thing that has probably saved her from a much more active manifestation of the disease is that strict attention which she has all along paid to the function of the bowels. The constant use of an appropriate aloin pill and frequent reference to salines never allowed any important derangement in this respect. Patient is one of those gouty people who value medical advice only in a theoretical way, and keeps in the wake of knowledge gained by self-instruction, but her experience has led her within recent years to give up taking soup and meat to lunch, and to lessen the amount of sweets and fruits. Various aches, pains, and tender feet are readily induced by any indiscretion, and such is by no means an infrequent occurrence.

What is the appropriate treatment? Owing to the long-standing nature of the disease, with the associated greater instability of tissue metabolism, and lessened power of resistance in the intestinal tract, the treatment will obviously differ in detail from any of those previously recorded. It may be well, in the first place, to give in full the diet which she has taken and finds well adapted to keep her free from gouty manifestations.

Breakfast.—Avoiding kidneys, steak, liver, and all made-up dishes, and only taking one solid, *e.g.* fish, of any kind (except salmon and fatty fishes which do not agree with her), one or two eggs, or bacon and egg; one breakfast cupful of freshly-made tea with not more than one piece of sugar. Toasted bread, Vienna rolls, or other bread (not too new or doughy), with butter. Marmalade or jam to be taken only in very small quantity, and even then not to be taken every day.

Lunch, to be mainly vegetables.—Selections from the following: tomatoes cooked or plain, macaroni dressed in various ways, salads, celery, cauliflower *au gratin.* Bread, brown bread, Vienna rolls, or any form of unsweetened biscuit; small piece of mild cheese, if desired. Small quantity of fruit, one of the following: an orange, raisins, apples, figs, dates, walnuts. Half an ounce of whisky in half a tumblerful of soda water.

Afternoon Tea.—One or two small cups of tea, with a very thin slice of bread and butter, or a piece of very light cake. Very little solid be taken, and especially no rich cakes.

Dinner.—The dinner to consist of three courses, to which fruit can be added, if not taken to lunch. The diet to be arranged on the following plan—

1	2	3	4	5
Soup	Fish	Soup	Fish	Soup
Meat	Meat	Entree[1]	Meat	Fish
Pudding	Savoury	Pudding	Pudding	Savoury
Fruit		Fruit		

Care has to be taken that on the nights when meat is taken the soup should be of a lighter character, *e.g.* clear brown, rice, or fish soup. Similarly on nights 2 and 4, the fish should be of the lighter kind, *e.g.* whiting, haddock, or sole. On the 5th night the soup can be richer, *e.g.* oxtail or kidney with a light fish, or the soup may be light and the fish more rich, *e.g.* turbot, halibut, or skate. No more than two vegetables are to be allowed, and then in sparing amount—a sauce to be reckoned as a vegetable. (Half an ounce of whisky in not more than half a tumblerful of water the only safe beverage for constant use.) Claret, champagne, and whisky are the only drinks for which patient has any regard. The puddings recommended are milk puddings of various kinds, stewed fruits made with small quantity of sugar.

Suet puddings only to be taken in sparing amount, and then only when the other courses are of the lighter nature. Jellies and creams can be taken sparingly, marangues (a special weakness) to be avoided. Bananas and strawberries are best avoided. Small cup of *café noir* prepared without sugar.

Are any medicinal remedies called for? As the patient is a sensible woman and fully appreciates the very small influence which drugs have exerted in her disease, none should be recommended with the exception of the constant use of a vegetable pill, and frequent use of the saline already referred to.

This case is a fair illustration of a not uncommon type in which the purely vegetarian diet, so eloquently advocated by Haig, is not applicable in treatment, and even if it were advisable there is not the remotest possibility of its being carried out. With regard to exercise, as the patient is of an active temperament, and has a considerable amount of muscular exercise in connection with her household duties and numerous outdoor interests, no further recommendations are called for. Further, it would be well for her to arrange to have her annual holiday occasionally at a health resort, where a full course of hydropathic treatment under medical directions will be useful in working off the effects of the occasional or frequent deviations from the paths of physiological righteousness, which may be regarded as a part of the clinical history of the disease; and in the intervals, and for the same purpose, an occasional course of diuretic remedies will be found of the greatest service.

Summary.

It may be well now to tabulate the principal points which should be kept in view in the general management of all cases.

1. When a hereditary tendency exists in children, habits of extreme sobriety in eating and drinking should be cultivated, and the diet should be mainly a milk, vegetarian, and light meat one.

2. When the disease is established there is no routine treatment, and the details will not be the same in any two cases. Attention should, however, be directed in turn to the following:—

   (*a*) The diet and state of the digestive tract.
   (*b*) The amount and nature of the exercise indulged in.
   (*c*) The functional activity of the skin and kidneys.
   (*d*) The state of the central nervous system.

3. The quantity of food should be determined by the amount of active exercise. Three meals per day only to be taken, and those to be of a simple character.

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[1] Sweetbread, tripe, chickens, rabbit.

4. When in doubt as to which set of food constituents to cut off, begin with the carbohydrates, and especially the saccharine substances. In not a few cases the latter may require to be completely cut off, and in every instance special care must be exercised in the admixture of carbohydrates with the fats and nitrogenous foodstuffs.

5. Thorough mastication of the food is all important, and strict attention to the evacuation of the bowels a necessity (with, in addition, an occasional saline).

6. *Fluids and Beverages.*—(*a*) Alcohol in any form is better avoided, except in subjects who are more or less habituated to its use, in which case the liquor taken should be *the one* which is known by the patient to be least detrimental to him. On no account should drinks be mixed. All sweet wines and malt liquors should be avoided, unless the patient is satisfied from careful observation that these are not prejudicial to him. The stimulant should be taken in a measured quantity and with meals.

(*b*) The free use of the alkaline table waters is to be commended, care being taken that an excessive amount of fluid is not taken with the meals.

(*c*) A tumblerful of hot water at night and an occasional course of mineral waters taken on an empty stomach are useful eliminants.

7. *Exercise.*—This should be adapted to the age and sex of the individual. Various forms of home gymnastics are useful in supplementing the course; in all cases care must be taken to avoid excessive muscular fatigue.

8. Attention to the excretory functions of the skin is of the first importance. This can be well done in the first instance at home by the daily morning bath, and the aid of a hot bath once a week or more frequently, followed by the stimulating effects of a cold spray. Flannels appropriate to the time of the year are to be worn. An occasional special course of hydrotherapeutics at a suitable health resort is desirable in all cases where such treatment is possible.

9. Medicinal remedies form the least important part of the treatment, but are beneficial in certain cases, more especially in those with a naturally weak digestive tract.

10. Owing to the profound influence of the mind upon the body, all the steps necessary to secure a state of mental rest (cessation of worry, change of air to a more bracing climate) must be advocated, and if these are not given effect to, the various other remedial measures may be of less avail.

LITERATURE.—The literature on gout is endless and a few references only will be given. Some of those mentioned contain full bibliographical lists to their respective dates.

General.—1. Sir A. GARROD. *Gout and Rheumatic Gout.*—2. EBSTEIN. *Die Natur und Behandlung der Gicht*, 1882; English translation (Burton) 1886, with literature.—3. Sir DYCE DUCKWORTH. *A Treatise on Gout*, 1889, with literature.—4. EWART. *Gout and Goutiness*, 1896, with literature.—5. LUFF. *The Pathology and Treatment of Gout*, 1898, with literature. —6. HAIG. *Uric Acid as a Factor in the Causation of Disease.*—7. Sir W. ROBERTS. "Gout" in Allbutt's *System of Medicine*, 1897.

8. **Special.**—By far the most recent and complete account of gout, especially in its therapeutic aspects, is Klinkowski's *Ernährungstherapie bei harnsaurer Diathese* (*Gicht*) *aus dem Handbuch der Ernährungstherapie*, Bd. II. 1899, with very full bibliography.—9. JONATHAN HUTCHINSON. Collected writings in the *Archives of Surgery*, 1894 to 1900.—10. CHALMERS WATSON. "Uric Acid and Gout. Some Points in the Physiology of Uric Acid," *Brit. Med. Jour.* Jan. 28, 1899; "Observations on General Metabolism and the Blood in Gout," *Brit. Med. Jour.* Jan. 6, 1900; "Metabolism in Gout, with Observations on the Effects of Salicylate of Soda and Nucleic Acid," *Jour. of Path. and Bacteriology*, Summer No. 1900. These three papers contain references to other important recent contributions.

Goundou. *See* NOSE.

Grain Poisoning. *See* TOXICOLOGY.

Green-Stick Fracture. *See* FRACTURES.

Groin, The.

Anatomy of	251	*Injuries of*	256
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THERE is no region of the body in which a greater variety of lesions is to be met with than the groin, and here, if anywhere, a precise anatomical knowledge is of the greatest importance for diagnosis. The groin is not, like the axilla, a very definite anatomical region, but it may be regarded as forming the junction of the abdomen, pelvis, and thigh; and as being the area which includes Scarpa's triangle, Poupart's ligament, and the parts immediately adjacent.[1]

The skin of the groin is thin and delicate in texture, and the effects of abnormal distension, such as phlegmasia alba dolens, are shown by the presence of striæ.

Owing to the thinness of the skin the groin is a region selected for the inunction of drugs, *e.g.* mercurial ointments and cod-liver oil.

I. ANATOMY OF THE GROIN

The bony landmarks to be recognised are the anterior superior spine of the ilium, the anterior fifth of the crest of the ilium, and the spine and crest of the pubes. In thin individuals, and in those who have become emaciated, additional bony parts can be made out, such as the anterior inferior spine of the ilium, the portion of bone between it and the anterior superior spine, the upper part of the rim of the acetabulum, the head of the femur, especially on flexion and rotation outwards of the thigh, and part of the horizontal ramus of the pubes. Poupart's ligament is the lower border of the aponeurosis of the external oblique muscle, and extends, with its convexity downwards, from the anterior superior spine of the ilium to the spine of the pubes; its reflection on to the pectinal line is known as Gimbernat's ligament. Poupart's ligament is relaxed when the thigh is flexed, adducted, and rotated inwards, and this fact should be borne in mind when the groin is being palpated. Between Poupart's ligament and the innominate bone are the structures which pass from the abdomen to the thigh, viz. psoas, iliacus, and pectineus muscles; the femoral artery and vein; and the external cutaneous, the anterior crural, and the crural branch of the genito-crural nerves. The inguinal canal, which contains the spermatic cord in the male and the round ligament of the uterus in the female, is about an inch and a half in length, has a direction downwards and inwards, and is parallel to and a little above Poupart's ligament; it commences at the internal abdominal ring, which is half an inch above the middle of Poupart's ligament, and ends at the external ring, which lies immediately above, and internal to the spine of the pubes; accordingly, the neck of an inguinal hernia lies internal to the spine of the pubes, while the neck of a femoral hernia lies external to this spine. The superficial fascia of the lower part of the abdomen and the upper part of the thigh is divisible into two layers, between which lie the superficial vessels and nerves. The superficial layer of this superficial fascia is thick and fatty, and in the male is continued over the penis and the outer surface of the cord to the scrotum, where it helps to form the dartos. The superficial vessels which lie between the two layers of the superficial fascia are the principal ones which are divided in herniotomy. The deep layer of the superficial fascia is a thin fibrous layer, and lies upon the fascia lata to which it is adherent at the lower margin of Poupart's ligament. Extravasated urine may pass from the scrotum to the abdomen between the attachment of the deep layer of the superficial fascia to the symphysis pubis and the pubic spine. The saphenous opening in the fascia lata lies an inch and a half below and external to the pubic spine, and is covered by the cribriform fascia, which is regarded by some anatomists as being a part of the deep layer of the superficial fascia. The deep fascia, or the fascia lata, forms a complete investment for the thigh, and its attachments in the groin are to the crest of the ilium, Poupart's ligament, the body of the pubes, and the descending ramus of the pubes; its iliac portion is that part of the fascia which lies external to the saphenous opening, at the lower margin of which it is continuous with the pubic portion. The crural or femoral sheath, a fascial arrangement which invests the femoral vessels as they pass below Poupart's ligament, consists in front of a continuation downwards of the fascia transversalis, and behind of the fascia iliacus. In addition, it is covered in front by the iliac portion of the fascia lata, and the pubic portion of the fascia lata lies behind it. The sheath is funnel-shaped, being broader above than below; it is divided into three compartments by two thin septa; the femoral artery occupies the outermost compartment, the femoral vein lies in the middle, and the innermost compartment—known as the femoral or crural canal—contains lymphatic vessels and a gland, and is the route which a femoral hernia takes in its journey from the abdomen to the thigh.

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[1] The general information which is given in this article will be supplemented in special articles, such as those upon "Hernia," "Aneurysm," etc.

The lymphatic glands in the region of the groin are numerous and important, for when they are enlarged, as they often are, as the result of conditions about to be mentioned, they increase the difficulties attending the diagnosis of tumours in this region. The following is Mr. Treves' lucid description of them:—

"They are divided into a superficial and deep set. The superficial set, averaging from ten to fifteen glands, is arranged in two clusters, one parallel and close to Poupart's ligament (the horizontal series), the other parallel and close to the long saphenous vein (the vertical series). The deep set, about four in number, is placed along the femoral vein, and occupies the crural canal.

"The inguinal glands receive the following lymphatics:—

"*Superficial vessels of lower limb* = vertical set of superficial glands.

"*Superficial vessels of lower half of abdomen* = middle glands of horizontal set.

"*Superficial vessels from outer surface of buttock* = external glands of horizontal set.

"*From inner surface of buttock* = internal glands of horizontal set (a few of these vessels go to the vertical glands).

"*Superficial vessels from external genitals* = horizontal glands, some few going to vertical set.

"*Superficial vessels of perineum* = vertical set.

"*Deep lymphatics of lower limb* = deep set of glands.

"The lymphatics that accompany the obturator, gluteal, and sciatic arteries, and the deep vessels of the penis, pass to the pelvis and have no concern with the inguinal glands."

Scarpa's triangle, which forms the lower portion of the groin, has for its base Poupart's ligament; its outer side, the sartorius muscle; its inner side, the inner border of the adductor longus; and its chief contents are the femoral artery and vein with their branches, including the origin of the profunda femoris artery and vein, the anterior crural nerve, and the termination of the internal saphenous vein.

Flexion of the thigh is a common condition, which may be temporary or permanent, partial or complete. In some cases it may be regarded as being a symptom and in others the result of disease. Though it will be fully dealt with in the article on "Diseases of the Hip-Joint," it may be mentioned in this place that temporary flexion of the thigh is often a symptom of value in the diagnosis of some of the diseases of the groin. It may be regarded as being Nature's method of diminishing pain about the hip joint. Flexion of the thigh, especially when combined with adduction and rotation inwards, relaxes the fascia lata, Poupart's ligament, and the abdominal muscles and fascia, and in consequence the pain of such conditions as synovitis of the hip-joint and strangulated inguinal or femoral hernia is relieved by reducing the pressure and allowing more room for the swelling. Extension of a flexed hip-joint should be carried out with caution, for cases are on record in which, during the operation, the skin and subcutaneous tissues were ruptured.

II. Diseases of the Groin

Skin diseases, as they affect the groin, merit no special attention; it may be noted, however, that pruritus genitalium not infrequently spreads from the scrotum or the labia to the groin, especially in very stout individuals. For further particulars about this and other affections of the skin the articles dealing with those subjects may be consulted.

Affections of the Lymphatic Glands.—Enlargement of the inguinal lymphatic glands is of frequent occurrence, the commonest causes being sepsis, tuberculosis, and syphilis. In most people, especially in males, and even in infants, lymphatic glands of the horizontal series are often found to be enlarged, as the result of frequent and trivial attacks of balanitis, or in consequence of the irritation of smegma preputii.

1. Septic inflammation of the glands is due, in the first instance, to the presence of a septic area in one of the regions—noted above—from which lymphatic vessels pass to one of the sets of lymphatic glands in the groin. Lymphangitis, of necessity, precedes this condition; its symptoms are—when the superficial lymphatics are affected—the presence of tender and somewhat cord-like, minute, red streaks in the situation of the lymphatic vessels, accompanied by some cellulitis of the adjacent tissues, which have an appearance almost indistinguishable from erysipelas. "Phlebitis closely resembles lymphangitis in its symptoms; a thrombosed vein forms a deeper-seated coarser cord than a similarly affected lymph vessel, the cutaneous redness is not so vivid, the pain is less acute, the general fever is not so intense, and the tendency to glandular involvement is less." Inflammation of the deep lymphatics is not easily differentiated from ordinary cellulitis.

The septic area from which the lymphatic vessels pass should be carefully sought for—a point which is often overlooked—and treated on general principles. If this lesion is detected early and efficiently treated, suppuration in the lymphatic glands may be prevented. If, however, suppuration has occurred, the glands should be laid freely open and stuffed with sterilised gauze. More harm than good may be done by making too small an incision in the gland, and the timid surgeon is apt to be over-cautious when operating upon glands in close proximity to large and important blood-vessels. The incision should be parallel to the principal vessels and nerves. Cellulitis of the surrounding tissues occasionally accompanies suppuration in the glands, and, if free incision of the glands does not arrest or cure the cellulitis, it is advisable to prolong the incision into the inflamed area. One of the commonest forms of suppuration in the inguinal glands is the result of a soft chancre on the penis, the treatment of which differs in no respect from that just mentioned. Before suppuration has occurred great relief may be given to the patient by applying fomentations of hot water, or hot lead and opium lotion; frequent changing of the fomentations enhances the value of this treatment.

2. A tuberculous condition of the inguinal lymphatic glands is not uncommon, resulting either as an infection from a tuberculous area, or as part of a general tuberculous infection of the glands. Several of the glands are usually affected, and they may remain enlarged for some time, and then disappear without any local treatment; or they may remain enlarged for a lengthened period without giving rise to any trouble; or they may undergo caseous degeneration, in which case the glands should be excised, or thoroughly curetted; or suppuration may take place in them, in which case incision being practically impossible or difficult to perform, curetting should be resorted to. General treatment, in the form of cod-liver oil, fresh air, and sunlight, should not be neglected.

3. Syphilitic affection of the inguinal glands is frequently met with, usually as a sequel to a hard chancre of the penis. Inguinal glands, when enlarged from this cause, do not, as a rule, suppurate if the patient rest; but if they do, a free incision and the application of some mercurial dressing, combined with constitutional treatment, is the indication.

In Hodgkin's disease or lymphadenoma, the lymphatic glands in the groin are enlarged, as are the lymphatic glands throughout the body generally. The glands form irregular and nodulated masses of various sizes, are either soft or firm, usually painless, and at first freely movable; later they may become adherent, but rarely caseate or suppurate. In the majority of cases no local treatment in the groin is necessary; for the general treatment of this condition the article on Hodgkin's disease should be consulted. (The diagnosis of this condition presents little difficulty; enlargement of the lymphatic glands, especially in the neck, the axilla, and the groin, associated with anæmia, is characteristic.)

Elephantiasis may be mentioned, for in those cases in which the leg and the thigh are affected, the groin shares in the general hypertrophy of the skin and subcutaneous tissues. Fistulæ in the groin are occasionally met with in this condition as the result of rupture of obstructed and distended lymphatic vessels.

Phlegmasia alba dolens, which is due to thrombosis of the femoral vein —and probably also of the iliac veins—produces extensive œdema of the lower extremity, resembles a mild case of elephantiasis in appearance, but could scarcely be mistaken for that condition.

The following is a list of the conditions which may be met with in the groin, each of which will be described in more detail in special articles (*q.v.*)—

Abscess and cellulitis; aneurysm; aneurysm with suppurating sac; arterio-venous aneurysm; varix of long saphenous vein; varix of femoral vein; phlebitis; phlegmasia alba dolens; enlargement of glands (adenitis; bubo); lymphadenoma; hernia,—inguinal, femoral, and obturator, with their varieties,—reducible, irreducible, and strangulated,—also the morbid conditions of the sac occasionally met with, viz.: hydrocele of the sac, and accumulation of ascitic fluid in the sac; various tumours—simple, sarcomatous, carcinomatous, and vascular; bursal tumours and cysts; undescended ovary and testicle, which may become inflamed in the inguinal canal; hydrocele, simple or diffuse, of the cord, and hæmatocele of the cord; psoas and iliac abscess; perityphlitic and perinephritic abscess; hip-joint disease; synovitis and suppuration in the hip-joint; dislocation, congenital and acquired, of the hip-joint; rider's bone, attributed to ossification of the tendon of the adductor longus or magnus as a consequence of injury.

The space allotted to this article prevents an elaborate description being given of the differential diagnosis of the numerous conditions just mentioned, and, therefore, it must suffice if a short account be given of the procedure to be adopted, and the points to be borne in mind, in making a diagnosis of the nature of a lump, swelling, or tumour in the groin.

1. The Age of the Patient.—Such conditions as aneurysm, malignant tumours, and rider's bone are unlikely to be met with in the very young, while in the aged tuberculous lesions are rare.

2. History of the Case.—A history of injury should be carefully inquired into; accurate information should be obtained about former illnesses and injuries and previous operations. The sudden appearance of the swelling would eliminate the different varieties of tumour, but might suggest hernia or aneurysm. The rapidity in growth of the swelling may vary between wide limits—a tumour, almost stationary as regards size, might be a hydrocele or one of the simple tumours, whereas rapid increase in growth might point to malignant disease, abscess, aneurysm, etc.

3. The Character of the Swelling.—If it disappears on manipulation it is unlikely to be anything but a hernia or a congenital hydrocele; if it is much reduced in size on pressure or by position it may be a varix, or a psoas, or iliac abscess. It is certain to be a varix if pressure above increases its size. Resonance on percussion would suggest an enterocele. The presence of fluctuation would eliminate a large class of swellings, *e.g.* the tumours proper, etc., but would indicate glandular and simple abscess and rarely aneurysm. Impulse on coughing is present in reducible and irreducible hernia, psoas abscess, and varix, and occasionally in cysts. A pulsating tumour is probably an aneurysm, or a vascular malignant growth, possibly a swelling, fluid or even solid, with pulsation communicated from the femoral artery. The presence of a thrill is characteristic of an aneurysm, or more rarely of a fluid tumour lying on the femoral artery. Pain is a prominent symptom in inflammatory conditions, in neuroma, and in tumours which press upon sensory nerves.

4. Signs of inflammation should also be noted. The swelling may be primarily an inflammatory one, *e.g.* abscess; or a pre-existing tumour, *e.g.* an aneurysm may become inflamed.

5. It is important to ascertain the existence of morbid conditions in any other part of the body which may have an important bearing on the lesion in the groin. Abdominal symptoms are, of course, a prominent feature in strangulated hernia and perityphlitic abscess, and their occurrence should be carefully noted. Thus, enlarged lymphatic glands in the neck and axilla, as well as in the groin, associated with anæmia, would confirm a diagnosis of Hodgkin's disease, and similarly a diagnosis of tuberculosis or syphilis might be made.

6. In the examination of the tumour it should be carefully ascertained if the tumour in the groin is confined to the groin, or if it extends into another region. Attention to this point might confirm a diagnosis of, *e.g.* psoas abscess.

III. Injuries of the Groin

Dislocations of the femur and fractures of the femur and of the pelvis are described in articles devoted to these subjects, and the possibility of suppuration following separation of the upper epiphysis of the femur should be borne in mind. Gunshot wounds are, especially during warfare, very common, and the nature and extent of the injury depends upon the size and shape of the projectile—an expanding bullet inflicting a very much more serious injury both upon the tissues and the bones than a non-expanding bullet; upon the velocity of the projectile—the greater the velocity the less serious the injury; and upon the distance which the projectile has to travel. Full details on these points will be found in the article on "Gunshot Wounds." When the artery is injured a traumatic aneurysm may result. Damage to the vein may cause an arterio-venous aneurysm, and if nerves, such as the anterior crural, are divided paralysis will result when the motor filaments are implicated, anæsthesia when the sensory filaments are implicated. The bone may escape injury, or it may be shattered. Every attempt must be made to save the limb, for few operations are attended with a higher mortality than amputation at the hip-joint, which such an injury would necessitate. Stabs and other punctured wounds may produce injuries similar to those just described, with the reservation that the subjacent bones can be, and usually are, damaged to only a slight extent.

Burns of the groin, more especially if deep, should be carefully treated, for unless the thigh is maintained extended by splints or extension during the healing process a cicatrix may result, causing almost incurable flexion of the thigh.

Great care should be taken to prevent the suppuration of wounds in the groin, for owing to the superficial position of the main vessels deep ulceration may result in secondary hæmorrhage.

Gums. *See* Jaws, Mouth.

Gunshot Wounds. *See* Wounds.

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Hæmatemesis.

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Hæmatemesis, or vomiting of blood from the stomach, must, in the first instance, be distinguished from the discharge by the mouth of blood derived from other sources. The commonest and most important of these is hæmoptysis; here the blood is of a bright scarlet colour, alkaline in reaction, frothy from admixture with air, and comes up with a cough, and for days afterwards the sputum, if there be any, is tinged with blood. It is, however, possible in profuse hæmoptysis for some of the blood to be swallowed and so give rise to secondary hæmatemesis. In epistaxis, or in fracture of the base of the skull, if the patient be unconscious, the blood may run down into the œsophagus and be swallowed, so that it enters the stomach, and when rejected is hæmatemesis. When the patient is conscious the blood may pass forward into the mouth and be spat out. Oozing from the gums, carious teeth, tonsils, or pharynx in various diseases may again sometimes imitate slight hæmatemesis. Anæmic girls not very infrequently speak of having brought up blood, which on further investigation proves to be very small in amount, and to occur in the early morning. In these cases the blood is probably provided by the naso-pharynx.

Feigned or hysterical hæmatemesis is sometimes due to industrious gum-sucking, to self-made wounds, or to extraneous blood or coloured fluids introduced into the mouth. The patient's manner and behaviour may arouse suspicion. The mouth and hands should be carefully examined for bleeding points, and the vomit submitted to microscopical and chemical tests for blood.

When blood is poured out into the œsophagus it may run down into the stomach, and when subsequently vomited is indistinguishable from ordinary hæmatemesis; this is particularly likely to occur when the source of the hæmorrhage is an ulcerated varicose vein near the lower end of the œsophagus. Sometimes when bleeding takes place from the walls of the œsophagus the blood does not enter the stomach, but wells up without any retching or vomiting. This is sometimes seen in ulcerated varicose veins, or in aortic aneurysm rupturing into the œsophagus; in both of these conditions the hæmorrhage may be very profuse. Small quantities of blood mixed with froth and mucus may come from the œsophagus in acute traumatic œsophagitis due to the ingestion of corrosive or irritant poisons, and is of course accompanied by great dysphagia. In rare instances blood and pus mixed together are hawked up from the œsophagus, and are due to one of the rare conditions, phlegmonous œsophagitis, or an abscess opening into the gullet. When simple ulceration of the mucous membrane of the œsophagus is present, vomiting from other causes may be streaked with blood.

Hæmorrhage is occasionally seen in the course of malignant disease of the œsophagus, which ulcerates early and produces symptoms of œsophageal obstruction. But it is noteworthy how extremely seldom carcinoma of the œsophagus ulcerates into the aorta, although their anatomical relationship is very intimate.

TRUE HÆMATEMESIS.—*Characters.*—The blood which is forcibly vomited up may be expelled through the nose as well as the mouth. Shortly before its onset the patient may feel distended, uncomfortable, faint, and become blanched.

As the vomited blood passes over the orifice of the larynx cough may be set up. This must be borne in mind, as otherwise this association of cough and blood may be regarded as proof of hæmoptysis. The cough follows the hæmatemesis, whereas in cases of copious hæmoptysis when some of the blood is swallowed the cough precedes the hæmatemesis.

Colour.—The blood is usually more or less altered in colour, being darker from the action of the gastric juice; on standing, however, it tends to pick up oxygen from the air, and so to become of a brighter hue; it is accordingly important that its naked-eyè appearances should be noticed at once. It will probably be acid in reaction and may be mixed with food.

When in copious hæmatemesis the blood is of an arterial character, rapid hæmorrhage from an artery exposed in a gastric ulcer has probably taken place. When on the other hand it is black, clotted, and copious, the bleeding probably depends on cirrhosis of the liver. Small "coffee ground" vomit is especially associated with carcinoma of the stomach; the blood is then very extensively acted upon by the gastric juice, and some question may arise as to whether the coffee ground vomit is blood or some article of food, drink, or medicine, such as coffee, porter, beef tea, charcoal, or even the mixture of tea and perchloride of iron (Bramwell). In such a case the hæmin test with salt and hydrochloric acid or the spectroscopic test should be employed, as being more reliable than microscopic examination, inasmuch as the corpuscles may be digested and destroyed.

Frequency.—Repeated large hæmorrhages may be due to ulcerated varicose veins at the lower end of the œsophagus in cirrhosis, to minute pore-like erosions of the gastric mucous membrane, or to gastric ulcer, and may so exhaust the patient as to be fatal. A single hæmatemesis is rarely fatal except when a large aneurysm bursts into the stomach or œsophagus—a rather rare event. It has, however, occurred in some cases of ulcerated œsophageal piles.

Small coffee ground vomiting, especially when repeated, points to carcinoma of the stomach; here vomiting is reflex and depends on the irritation of the growth. Any blood that happens to be on the stomach is accordingly brought up with the other gastric contents; in large hæmatemesis, as in gastric ulcer, the vomiting is the result of stimulation of the gastric muscular walls by the distension of the organ, and is comparable to the action of an enema on the lower bowel.

In very rare instances hæmorrhages may recur at intervals over a period of years with good health in between; this has been observed in splenic anæmia (Osler), and exceptionally in cirrhosis from varicose veins at the lower end of the œsophagus (Garland).

Causes and Source.—The blood may come from the œsophagus or duodenum as well as from the stomach itself.

Varicose œsophageal veins are generally part of the compensatory venous anastomosis set up in hepatic cirrhosis, but they may also develop in extensive malignant infiltration, for example, in secondary melanotic sarcoma of the liver, where this induces portal obstruction, or they may be idiopathic, viz. without any causative lesion.

As a result of chronic inflammation the varicose veins tend to become adherent to the mucous membrane, and ulceration may follow with profuse or even fatal hæmorrhage. This event may come on without any previous symptoms of ill health, the underlying cirrhosis being quite latent.

Duodenal ulcer may give rise to regurgitation of blood into the stomach and hæmatemesis. Ordinary duodenal ulcers are found in the first part of the duodenum; hæmorrhage may also occur from the ulceration and passage of gall-stones into the duodenum. An abdominal aneurysm rupturing into the duodenum has been known to give rise to fatal hæmatemesis, while an hepatic aneurysm bursting into the bile duct may also be a cause of hæmatemesis, though melæna alone is more often recorded.

In the stomach the lesions that give rise to hæmatemesis may be divided into

(1) Gross lesions.
(2) More minute lesions of the mucous membrane.

(i.) *Gross Lesions.*—Gastric ulcer is more frequent in anæmic young women, and is then commonest near the pylorus. It also occurs in men at a more advanced age, and is often more extensive and firmly adherent to neighbouring viscera than in the first category; it may be situated in any part of the stomach. Repeated and large hæmorrhages may occur from minute abrasions of the gastric mucous membrane, "pore-like erosions," or "exulceratio simplex."

Small ulcers may supervene in chronic engorgements of the stomach as the result of localised hæmorrhages into the mucous coat. The ulceration may open up a vessel and give rise to profuse hæmatemesis, and from their minute size they may easily be overlooked. Possibly some cases of fatal hæmatemesis, where the stomach has been described as perfectly healthy, are of this category. These small ulcers are often seen near the cardiac orifice; in the passive congestion due to cirrhosis these ulcers usually open up a vein, while in that due to cardiac disease an artery is eroded (S. Fenwick).

Varicose veins of the stomach are much rarer than in the lower part of the œsophagus, but may give rise to severe hæmatemesis.

Other rare causes of hæmatemesis are aneurysms of the gastric, splenic, or hepatic arteries opening into the stomach. Carcinoma of the stomach may arise at the cardiac orifice, at the cardiac end, at the pylorus, or may involve the whole of the organ. The hæmorrhage is usually slight, rarely profuse, and, as already pointed out, the blood is apt to be retained in the stomach, and as a result of the action of the gastric juice become black or "coffee ground" in appearance. In very rare instances a malignant growth may invade the stomach from without, and give rise to hæmatemesis. The writer has seen this in a case of carcinoma of the left suprarenal body.

(ii.) More minute lesions of the gastric mucous membrane occur in acute gastritis. This may be of local origin, and follow the ingestion of toxic fluids, such as large quantities of spirit, acids, phosphorus, or other poisons.

In chronic enlargement of the stomach depending on the backward pressure of heart disease, hepatic cirrhosis, thrombosis of the portal vein, or compression of the gastric veins by adhesions, inflammatory and degenerative changes in the mucous membrane of the stomach are readily set up, with the result that oozing of blood follows. In chronic engorgement of the stomach the further change of gastritis may be readily set up by swallowing the pus and micro-organisms present in dental caries and pyorrhœa alveolaris. In cirrhosis of the liver it is generally assumed that hæmatemesis may be due to a widespread venous or capillary oozing, but it is probable that in addition to the venous stasis some degenerative changes in the mucous membrane are necessary to allow of the extravasation of blood. In splenic anæmia, however, hæmatemesis has been explained by Osler as being purely mechanical and due to venous engorgement.

Changes in the gastric mucous membrane may be part of a general condition, and may be secondary to severe hæmic infections or intoxications, such as yellow fever, icterus gravis, acute yellow atrophy of the liver, and the hæmorrhagic or malignant forms of the specific fevers. In these, and sometimes in pyæmic and septicæmic states, blood may be extravasated in varying amounts into the stomach. In diphtheria marked degeneration of the gastric mucous membrane is met with, and blood may be found in the vomit in cases that are not of the hæmorrhagic type.

Hæmatemesis is very rare in typhoid fever, but has been known to occur and is correlated with the rare lesion of typhoid ulcers in that organ. In a few instances pneumonia is accompanied by hæmatemesis, due to secondary ulceration in the stomach or duodenum. Hæmatemesis may occur in hæmorrhagic states, such as purpura hæmorrhagica, leukæmia, pernicious anæmia, and hæmophilia. It has also been observed in association with urticaria, and the two are doubtless manifestations of the same underlying blood state.

Hæmatemesis is said to represent menstruation. It is, however, quite possible that the explanation of some of these cases of vicarious menstruation is that there is a chronic gastric ulcer which periodically bleeds, while in others the hæmorrhage may have been feigned. Vicarious menstruation was recognised by Sir Thomas Watson, but at the present time most practising physicians will endorse the late Dr. J. Matthews Duncan's dictum—"I have all my life been on the look-out for it" (vicarious menstruation), "but I have never seen an example, and do not expect to do so."

Diagnosis.—Sudden hæmatemesis without any history of previous illness is in a middle-aged patient most likely to be due to latent cirrhosis of the liver. If the liver and spleen are found to be enlarged, and the individual's habits are known to be alcoholic, the diagnosis is strengthened. If the sudden hæmorrhage is accompanied by the rapid development of ascites, thrombosis of the portal vein should be thought of. Great enlargement of the spleen would point to spleno-medullary leukæmia or splenic anæmia, and a blood examination should be made to elucidate the diagnosis.

A large hæmatemesis may occur from acute gastritis due to recent alcoholic excess, and the question will arise as to whether there is latent cirrhosis as well. The absence of any evidence of cirrhosis and the occurrence of hæmatemesis in a young man are in favour of alcoholic gastritis.

If there has been dyspepsia for some time before the onset of hæmatemesis the cause may be gastric ulcer, carcinoma of the stomach, cirrhosis, or duodenal ulcer. If the patient is a young anæmic woman and the hæmorrhage is copious, the probabilities are strongly in favour of a gastric ulcer; this will be supported by a history of pain, which coming on directly after food is relieved by vomiting, and by finding a point of maximum tenderness on deep pressure over the stomach; with simple pore-like erosions of the gastric mucous membrane, tenderness, however, may be absent, though the hæmatemesis may be frequently repeated and severe. Examination of the vomited matters, apart from hæmatemesis, will show an excess of hydrochloric acid in gastric ulcer. Melæna follows hæmatemesis. In very rare cases of hour-glass stomach with an ulcer in the distal pouch, melæna may occur without hæmatemesis. In other cases where the hæmorrhage is slight melæna alone may be noted.

Gastric ulcer in men is met with later in life than in the female sex; it is accompanied by more pain, gives rise to anæmia, and may be associated with arterio-sclerosis. Care must be taken to eliminate carcinoma, cirrhosis, and duodenal ulcer.

It should be remembered that carcinoma may develop in the site of a chronic gastric ulcer, and that as this change takes place a corresponding modification in the symptoms may be observed.

In carcinoma of the stomach the vomited blood is black, resembles "coffee grounds," and is usually small in quantity, so that melæna is not noticed. If tested, the vomit will probably be found not to contain hydrochloric acid. A most important point in the diagnosis of gastric carcinoma is the presence of a tumour in the stomach or in its neighbourhood. Thus a tumour may be felt near or at the umbilicus, in the line of the falciform ligament, or on the surface of the liver. The age of the patient has a very definite bearing; ulcer is common in early life, while carcinoma is rare.

Hæmatemesis in cirrhosis may be preceded by chronic dyspepsia, but the symptoms are not so severe as in ulcer or carcinoma, and tenderness, if present, is general and much less than the localised pain elicited on pressing over a gastric ulcer.

In duodenal ulcer, the pain and dyspepsia should come on two hours after food, *i.e.* when the pylorus allows the contents of the stomach to pass into the duodenum, tenderness is more to the right of the middle line than in gastric ulcer, and the patient is nearly always a man. Melæna may precede hæmatemesis, or even occur without hæmatemesis.

When hæmatemesis is associated with jaundice, acute yellow atrophy of the liver, phosphorus poisoning, or cholæmia from the continued effects of obstructive jaundice should be thought of.

Hæmatemesis with fever suggests some severe infection, while hæmorrhage elsewhere points to a blood condition such as purpura, the malignant or hæmorrhagic forms of the specific fevers, pernicious anæmia, etc.

Prognosis.—Hæmatemesis is rarely immediately fatal; when it is so in cirrhosis, the first hæmorrhage is in a considerable proportion followed by death (Preble). The rupture of an abdominal aneurysm is of course likely to be fatal at once, while the copious hæmorrhage from an ulcerated œsophageal varix, or from a large artery like the splenic opened by a gastric ulcer, is much more prone to be followed by immediate grave results than the small hæmorrhages of malignant disease. When large hæmorrhages occur at short intervals, death may occur from exhaustion; in such cases the stomach should be opened and the bleeding point surgically treated.

Hæmatemesis in yellow fever and in the malignant forms of the specific fevers is of course indicative of the worst prognosis, as showing the virulence of the primary disease, while blood in the vomit of cases of diphtheria without hæmorrhages elsewhere is a very ominous sign, inasmuch as death from cardiac failure is very likely to follow, the heart muscle, like the stomach, having undergone toxic degeneration.

The more remote prognosis is that of the morbid lesion responsible for the hæmatemesis; thus the small "coffee ground" vomit of malignant disease is of infinitely more serious import than the copious hæmatemesis of gastric ulcer.

Treatment.—The patient should be kept absolutely at rest, and the head flat so as to prevent syncope. Mental anxiety may be relieved by the hypodermic injection of morphia.

Nothing should be given by the mouth, and thirst, which after a large hæmorrhage may be urgent, should be relieved by enemata of 8 to 10 oz. of water every four hours.

Various styptics may be given by the mouth for their local action on the stomach, such as acetate of lead 2 grs. every three hours; gallic acid grs. x., combined with dilute sulphuric acid ♏x.; opium in various forms, oil of turpentine ♏xx. every six hours. I have found half-drachm doses of Ruspini's styptic in an ounce of water act well; suprarenal extract by the mouth has recently been suggested as a local styptic. In order to increase the coagulating power of the blood, calcium chloride in full doses (grs. xxx.) every two hours for a few doses may be tried. The hypodermic injection of gelatine has been employed with the same object.

The hypodermic injection of ergotin or digitalin is not advisable. The local application of an ice-bag over the stomach has been widely recommended, does not appear to do any harm, and may do good in several ways.

In cases with very severe anæmia and collapse, intravenous transfusion of saline solution should be resorted to. When large hæmorrhages are repeated at short intervals, or small hæmorrhages occur very frequently, the abdomen should be opened, the stomach incised, and the mucous membrane examined, any ulcer or pore-like erosion should be treated surgically.

In repeated hæmatemesis of splenic anæmia splenectomy has been known to prove a curative measure.

It is advisable to give a purge, blue pill, and haustus sennæ two days after hæmatemesis due to cirrhosis; in cases of ulcer it is wisest to be content with enemata.

The dietetic and further treatment varies with the cause; thus in gastric ulcer, rectal feeding should be the rule for about two weeks, whereas in cirrhosis liquid food may be cautiously given, provided there is no recurrence two or three days after the hæmatemesis. In the smaller hæmatemesis of malignant disease and that symptomatic of fevers and hæmorrhagic disease, food should be given earlier.

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Hæmatoma Auris.

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Conditions of Occurrence.—Hæmatoma auris, otherwise known as othæmatoma, or the insane ear, occurs in its typical form in persons of unsound mind. A few instances have been recorded of the occurrence of an apparently similar condition unassociated with insanity, but in all probability either these cases are purely traumatic in origin, and therefore essentially dissimilar, or the patients are of degenerate habits, and mentally not far removed from the overtly insane. The broad fact remains that it is in lunatic asylums that the great majority of such cases are found. Even among the insane, however, only a certain small proportion suffer, estimated by Lennox Browne at 2·24 per cent, by Campbell at 2·23 per cent, and by Langdon Down as high as 3·6 per cent of male congenital idiots. It may be noted that these figures over-estimate rather than under-estimate the average frequency in most modern asylums. The number of men affected is relatively greater than the number of women. A somewhat peculiar and inexplicable fact is that the left ear is much more commonly involved than the right. Moreover, when both ears are implicated, as they are in about one-third of the total number of cases, it is the left which is usually first affected.

There is no particular form of insanity exclusively associated with othæmatoma. It has been found in all the more common varieties, but there is some difference of opinion concerning the types with which it is especially correlated. Clouston states that it is very common in general paralysis, and is sometimes seen in bad cases of mania of the chronic variety, sometimes in chronic epileptics, occasionally in agitated and convulsive melancholia, and rarely in dementia. Lennox Browne found that it occurred for the most part in patients subject to attacks of a violent and paroxysmal character. Out of thirty-two cases collected by him, seventeen suffered from mania acute or chronic, eight from general paralysis, five from epilepsy, and only two from dementia (not epileptic). Langdon Down, in noting its frequency in male congenital idiots, states that it is rare in female idiots and in accidental idiocy, that it may be found in idiocy arising from developmental causes, but almost never when the cause is operative in the early days of post-uterine life. The majority of idiots whom he observed to be subject to othæmatomata were also epileptic, and in all the element of excitement was excessive.

Clinical Features.—Othæmatoma of the insane first appears as a swelling in the external ear, and is almost invariably limited to the cartilaginous portion. It starts most commonly in the helix, and may then be bounded by the line of the anti-helix, or may extend more widely. Less frequently it begins elsewhere, as in the concha or fossa triangularis, and only very rarely in the external auditory meatus. In its earliest stage there occurs a small, somewhat red or livid, tense, cystic swelling which tends to increase more or less rapidly, until in the course of a few days it attains the size of a hazel nut or a hen's egg. The surface may be quite smooth, or may retain traces of the ridges in the fossa of the helix. Some irritation and discomfort are usually present, but seldom actual pain, and there are no subjective auditory symptoms such as deafness or tinnitus unless the external meatus is implicated. If proper treatment be withheld the cyst continues to enlarge, and may ultimately rupture, discharging a bloody or gelatinous fluid. After a week or two, unless the process is interrupted by fresh hæmorrhages, the cyst contents begin to be absorbed, the cyst wall undergoes shrinkage, and ultimately the auricle becomes greatly atrophied, puckered, and permanently distorted. In exceptional cases, quite apart from any surgical interference, there may occur accidental inoculation with pyogenic microbes, suppuration sets in, and the cyst contents become purulent. Such a condition may, though rarely under antiseptic treatment, lead to extensive necrosis and gangrene of the external ear.

It is worthy of note that the prompt application of blistering fluid to the site of the swelling may, and very often does, not only arrest its further course, but greatly diminishes the amount of the subsequent deformity.

Morbid Anatomy.—Examination of a recent case of othæmatoma of the insane reveals the presence of an irregular cystic cavity situate usually between the anterior perichondrium and the cartilage of the pinna, as if these structures had been torn asunder by an effusion of blood. The cyst contents present great diversity of appearance, being sometimes dark red in colour and watery in consistence, sometimes pale yellow, translucent, and gelatinous. The effusion for the most part consists of blood in various stages of coagulation and decolorisation. The wall of the cyst shows an irregular lining of granulation tissue with newly-formed blood-vessels, which are often extensively degenerated. The adjacent cartilage shows numerous patches of degeneration which are described below, and which in all probability represent the essential preliminary lesion. In all ordinary cases which have not undergone accidental inoculation there is an entire absence of micro-organisms both from the cyst cavity and from the diseased cartilage.

As the condition persists, an increased formation of granulation tissue lines the interior of the cyst wall, organises and absorbs the effusion, and ultimately, by its further development into dense fibrous tissue, produces the extreme permanent contraction and distortion so characteristic of long-standing untreated cases.

Etiology.—Widely divergent views have been expressed regarding the causation and mode of origin of this condition, but only a brief outline of the more important theories need be given here. Most observers would probably concur in the statement that there is in the first instance some local predisposing condition under the influence of which the insane are rendered more prone than those in full mental health, and in the second place some local determining factor to the operation of which the actual onset is due.

The nature of the local predisposing cause was first suggested in 1848 by Fischer, who discovered the presence of cysts in the ear cartilages of the insane, and ascribed the occurrence of hæmatomata to primary hæmorrhage into them. Subsequently Virchow in 1863, Pareidt in 1864, L. Meyer in 1865, and Tischkow in 1891, confirmed and elaborated these earlier observations. More recently Ford Robertson in 1896 completed an extensive and careful examination of the ear cartilages both in the sane and in the insane, and found that in each series of cases there were present degenerative changes similar in kind but varying greatly in degree. The earliest evidence of degeneration is observed in the cartilage cells, which throughout areas of varying size became vacuolated and later disintegrated. The yellow elastic fibres in the same area are broken up into minute droplets and finally disappear. The central portion of the degenerated patch, more especially if large, tends to undergo liquefaction, thus leading to the formation of a small cyst. Vascular granulation tissue then replaces the degenerated patch of cartilage or lines the wall of the small cyst. In fifty cartilages from presumably sane patients, Ford Robertson found that no fewer than forty-eight showed some degenerative change, but he states that in the majority of these it consisted merely in the loss of the elastic fibres, and in slight degeneration of the cartilage cells in exceedingly minute areas. Granulation tissue replaced small areas of cartilage in eleven cases; but only in eight cases had the process advanced to the formation of a cyst, and in no case had vascularisation of the cyst wall occurred. In fifty cartilages from the insane there were as before only two cases in which degenerative changes were entirely absent, but in the number of areas affected and in the extent of tissue involved there was a contrast of the most marked kind. Cyst formation had occurred in no fewer than thirty cases, and in eight of these the cyst wall had become vascularised. There was, in short, abundant evidence of profound nutritional changes of a degenerative nature in the ear cartilages of the insane as compared with those of the mentally sound.

Moreover, the young vessels in the newly-formed granulation tissue were themselves extensively degenerated, and thereby rendered unduly liable to rupture.

Many other explanations have been offered of the predisposition of the insane to othæmatoma, but these are rather tentative suggestions than statements of actual fact. Thus Alexander Robertson considered that a local vascular engorgement produced by disturbance of the cervical sympathetic system was the essential predisposing cause, and in this hypothesis he was supported by Pietersen and others. An obvious comment is that in exophthalmic goitre—a disease in which extreme disturbance of the cervical sympathetic system is undoubtedly present—there is no special predisposition to othæmatoma. Even such conditions as a blood dyscrasia, a lesion of the restiform bodies, etc., have been alleged to be the chief causal agents.

In all probability the actual effusion of blood from the diseased new vessels in the degenerative ear cartilage is determined by some insignificant traumatism. A slight accidental blow, or even the mere friction of the ear between the head and the pillow in a restless patient would suffice. It is quite unnecessary to assume that there has invariably been rough or careless treatment or a severe local contusion, and it is quite erroneous to maintain, as has been done by several writers, that all cases of othæmatoma are purely traumatic in origin.

Onset apart from Insanity.—Cases of hæmatoma auris in persons who are not under treatment for mental disease do undoubtedly occur, and may be included in one of two categories. If the subjects are in full mental health with undegenerated ear cartilages, the condition is unknown apart from severe local injury, *e.g.* fracture of the cartilage and laceration of healthy vessels. It has been known to occur at football, boxing, wrestling, or in other circumstances where the ears are exposed to considerable violence, but even then it is exceedingly rare. Moreover, the frequency with which some children receive a violent box on the ear without any damage of this nature, shows that, when the vessels and cartilages are healthy, hæmorrhage is not readily produced. Another class of patients, however, though not confined in asylums for the insane, includes those of degenerate mental powers, alcoholic habits, etc. From the researches of Ford Robertson on the ear cartilages of the presumably sane it is permissible to infer that in such cases considerable degeneration of the cartilage is also present. In other words, these represent a group of transitional cases in which the degeneration is not so advanced as it is in the insane, but yet sufficiently marked to allow hæmorrhage to occur from an injury of only moderate severity. The literature of the subject abounds with cases which support this view.

Relation to Microbic Infection.—The earliest reference to the infective origin of othæmatoma was made in 1846 by Leubuscher, who applied the term erysipelas auriculæ to the condition. Subsequently, from 1892 to 1896, Pellizzi published the results of a bacteriological research, from which he concluded that the ordinary hæmatoma auris of the insane is due to a streptococcus indistinguishable from the streptococcus pyogenes vel erysipelatis. Vassale, quoted by Pellizzi, failed to find any organism in one case examined by him. In 1894 Goodall stated that out of seven cases five showed the presence of the pyogenic staphylococci, both aureus and albus, while two were absolutely sterile. The attention of the writer was directed to this subject in 1895, and two typical cases of othæmatoma were examined. In each the cyst contents were at first perfectly free from micro-organisms, but in one case accidental inoculation subsequently occurred, the cyst contents became purulent, and then, and not till then, streptococci were abundantly present. Pieces of ear cartilage showing the early degenerative changes which precede the onset of an actual hæmatoma were also investigated, but no bacteria were detected.

The organisms so frequently present must be either the cause or an accident of the condition. In the former case, either they must have the power of producing those early degenerative changes in the cartilage which precede the formation of the hæmatoma, or they must by their presence determine the occurrence of the hæmatoma from a degenerated patch already present, or from healthy cartilage. There is, however, absolutely no proof that micro-organisms are at all related to simple cartilage degeneration. Hence, if the presence of micro-organisms is not to be considered purely accidental, it must be shown that they are the direct cause of the hæmatoma. This is the position for which Pellizzi contends, and which Goodall is inclined to support, but it is quite untenable, for the following reasons:—(1) The cases quoted by Pellizzi, Goodall, and the writer, show that no single organism can be regarded as the specific cause, since at least three different varieties have been found. (2) The results of Vassale, Goodall, and the writer, further show that organisms are by no means constantly present, but that there is a considerable proportion of cases which, when examined with all due care, give purely negative results. (3) By inoculation of pure cultures Pellizzi has consistently failed to reproduce the condition, causing simply inflammation and suppuration in varying degrees of severity. (4) The organisms hitherto discovered are all identical with well-known pyogenic forms which have never been found to produce any condition analogous to hæmatoma auris.

The conclusion, therefore, to which the evidence most strongly points is, that the relation of micro-organisms to othæmatoma is purely accidental. It is not the presence of organisms that determines the formation of the hæmatoma, but, on the contrary, it is the hæmatoma that affords a subsequent nidus for organisms. Nor is it difficult to understand how such access may be obtained. Any subcutaneous effusion is more or less liable to become inoculated, and, in the insane, this liability is considerably increased, more especially when such an exposed part as the ear is affected. For, owing to the carelessness, or restlessness, or actual violence of the patient, or owing to his fingering the damaged part, there may readily be produced an abrasion which may become the channel of infection.

Prognosis.—The result to the auricle affected depends chiefly on the promptitude with which suitable treatment is applied. If the condition be permitted to develop to its full extent without any remedial measures being undertaken, the subsequent deformity is usually extreme; whereas the early application of blistering fluid usually succeeds in arresting the progress of the effusion, and in very greatly diminishing the amount of permanent deformity.

The prognostic value of othæmatoma as regards the progress of the mental malady is important. Its presence is indicative of widespread and profound nutritional changes, and a corresponding extent and degree of cerebral degeneration. The prospect of ultimate recovery is, therefore, very slight. Clouston states that he has "seen only four or five cases perfectly recover out of over eighty who had fully developed hæmatoma auris, and four others who made partial recoveries after slight threatenings of hæmatoma auris which might not have developed fully or were stopped by blistering fluid."

Treatment.—The most effective means of treatment is that first suggested by Dr. Hearder, and indicated in the preceding section. It consists in the application of blistering fluid to the auricular surfaces on the earliest signs of any swelling from the commencing hæmatoma. Its success depends upon the promptitude with which the fluid is painted on, and the failure of the method is usually proportionate to the delay. If, as sometimes happens, the cyst becomes accidentally inoculated with pyogenic organisms, it must be freely incised, and the purulent contents washed out with antiseptic solutions. Except in these circumstances, however, surgical interference should be avoided as tending more to aggravate than to improve the condition.

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Hæmaturia.—According to its etymological sense the term should be restricted to cases in which blood is effused from the vessels of the kidneys, ureters, or bladder, and discharged along with the urine, excluding from the definition urethral hæmorrhage, in which the blood escapes by drops or flows in a continuous stream from the orifice of the urethra, and which is not properly a mictus cruentus.

The appearance of blood in the urine during micturition is a symptom of a large number of different lesions, and, as seen in surgical practice, may have its origin in any of the divisions of the urinary tract. Hæmaturia may be due to (*a*) lesions of the renal parenchyma or the pelvis; (*b*) disease of the ureters; (*c*) disease of the bladder; (*d*) disease of the prostate; (*e*) disease of the urethra; and (*f*) disease of the testicles.

The pathological conditions which are associated with the appearance of blood in the urine are therefore very various and numerous. The great majority come under the cognisance of the surgeon, but there are some which belong strictly to medical practice. For example, hæmaturia may be an accompaniment of hæmophilia, septicæmia, typhus, enteric, or malarial fevers, small-pox, scurvy, or purpura; or it may be the direct consequence of poisonous agents, such as cantharides, alcohol, turpentine, phosphorus, and arsenic.

There is an intermittent form of hæmaturia which has been described under the term "malarial hæmaturia," more common in men than in women, and differs from ordinary "paroxysmal hæmaturia" in the greater regularity of the attacks, in that it is met with in certain districts only, and in that it is associated with the presence of malarial organisms in the blood.

What is called "endemic hæmaturia" depends upon the presence of a parasite, the *Bilharzia hæmatobia.* This form of the disease is met with in Egypt, Natal, Cape Colony, Mauritius, and in Brazil, and a somewhat similar hæmaturia prevails in India, due to the presence of the *Filaria sanguinis hominis.* These conditions only require to be mentioned, as they are rarely met with by practitioners at home. (*See* "Parasites.")

Tests.—Tests for blood in urine may be divided into three classes:—

(*a*) *Microscopic.*—When the urine is allowed to stand the corpuscles and colouring matter may fall as a deposit, when they are easily recognised by the microscopic examination. This is most readily seen when the urine is acid or neutral; but when it is alkaline the colouring matter is liable to be dissolved out of the corpuscles, and these by imbibition of fluid become distended and appear as almost colourless spheres instead of flat discs. Their characteristic appearance is therefore lost. Occasionally the edges may become serrated from shrivelling of the corpuscles.

(*b*) *Spectroscopic* examination affords a very delicate test. Blood even in very minute quantities gives the characteristic absorption bands of hæmoglobin in the yellow and green between the D and E lines.

(*c*) *Chemical* tests may be also employed. (1) The addition of carbolic acid to a urine containing blood causes coagulation of any albumin which may be present, and also changes the colour of the fluid to a peculiar reddish tinge. (2) By boiling the urine with caustic soda and allowing it to stand, a brick-red precipitate is thrown down. (3) When a little glacial acetic acid and a crystal of common salt is added to urine containing blood, Teichmann's crystals are deposited. (4) When a couple of drops of tincture of guaiacum and half a drachm of "ozonic ether" are added to a drachm of the urine in a test-tube and the whole shaken, the ether dissolves the resin which has been precipitated, and after a few seconds carries it to the surface of the urine. At the line of union between the "ozonic ether" and the fluid lying underneath it, if blood be present, a blue coloration appears.

FALLACIES.—Red urine is sometimes passed by patients who have taken sulphonal in large doses or for a long period. The excretion may also be rendered red or black from the admixture of various pigments derived from articles of diet or medicine. Rhubarb, beetroot, strawberries, sorrel, prickly pear, and logwood may impart a red colour. Senna, salicylic acid, and carbolic acid may also cause coloration.

In microscopic examinations, globular vegetable spores may simulate blood corpuscles. In using the spectroscope it must be remembered that logwood gives a spectrum very similar to hæmoglobin, and in employing the guaiacum test it must be borne in mind that saliva gives the same reaction as blood.

Differential Diagnosis of the Source of the Hæmorrhage.—The methods employed may be classified as follows:—

Firstly, by observing (*a*) the physical characters of the urine and of the blood-clot; (*b*) the admixture of other deposits with the blood; (*c*) the time at which the blood appears in the stream; (*d*) the frequency and duration of attacks; and (*e*) the effects of movements and exercise, or of complete rest, in the course of an attack. Secondly, by collecting the urine separately from the two ureters, or by observing blood escaping from the orifice of a ureter. Thirdly, by carefully considering all the other accompanying objective and subjective phenomena.

I. (*a*) *Physical Examination of the Urine and the Circumstances connected with the Presence of Blood in the Evacuation.*—The following questions must be inquired into and answered:—What is the colour of the blood? Is it intimately mixed with the urine, and is the whole stream equally colourised? Is the quantity of blood augmented by exercise or diminished by rest? Have clots been seen, and what is their form? Are there tube-casts or blood-casts to be discovered, or are there other deposits found in the urine? Are the attacks frequently repeated and of short duration, or are they protracted?

The colour imparted to the urine by the addition of blood varies greatly in intensity and shade—from a pale rose colour to a bright red, like red currant syrup mixed in water, to a dark red, to a colour simulating porter, or to a brown coloration not unlike coffee. As a general rule, however, it may be stated that the nearer the source of hæmorrhage is to the external orifice of the urethra the less is the blood altered in appearance.

When the hæmaturia is of *vesical origin*, the first quantity of urine passed may present a normal appearance, especially if the patient has been for some time at rest in a recumbent posture, and the urine is passed while the patient is in bed; but in such circumstances, as the bladder empties itself, the urine gradually becomes more and more tinged, till finally the last drops evacuated may be almost pure blood. This is to be especially noted in cases of tumour of the bladder and of vesical calculus, also in passive hyperæmia of the bladder. As a general rule, in hæmorrhage from the bladder the urine is mixed with a considerable quantity of mucus, and is of a bright florid colour; but, if long pent up in the viscus, it may assume a dark colour dependent upon the reaction of the urine, which in such cases is usually alkaline. When due to vesical calculus the hæmorrhage is increased by exertion, and in cases of tumour of the bladder the bleeding is always worse after an attempt has been made to examine the bladder by instruments.

When the blood is from the *kidneys* or the *ureters* it is generally intimately mixed with the urine, to which it imparts a dark smoky colour, unless when the blood issues in considerable quantity from the pelvis of the kidney, and flows rapidly along the ureter, distends the bladder, and escapes without undergoing much change.

Blood having escaped from the vessels, its fibrous constituents may coagulate into firm clots, which during their formation may entangle some of the histological elements of the structures in which they lie, or by their firm appearance, form, or bulk may indicate the source of the hæmorrhage. Clots are seldom noticed in the urine unless in cases of abundant hæmorrhage; they may be few in number or numerous; generally they are soft in consistence, and when small in size they are easily dissolved in the urine, and consequently, if not looked for immediately after micturition, they may be lost. The form of the clot may be ovoid, or may be long and worm-shaped, or may resemble in appearance a well-gorged leech. As to colour they are generally dark red, but may vary considerably, sometimes being black, bright red, or they may become partly or wholly discoloured—spotted red in a grayish background, or entirely gray. When the clot is large the hæmorrhage is usually, but not invariably, from the bladder; the clots may be so huge that they cannot pass along the urethra without being broken up either by the repeated contraction of the bladder or by means of instruments. Rounded clots corresponding to the diameter of the ureter may escape, or leech-like ovoid casts may be taken of the first part of the urethra, or long bougie-like coagula from its anterior part. While the presence of considerable clots is usually, though not always, conclusively against the idea of the hæmorrhage being from the secreting substance of the kidney, the absence of clots visible to the eye proves nothing. If the passage of a long worm-shaped blood-clot is preceded by a temporary cessation of the hæmaturia, and its escape followed by a recurrence of the bleeding, this proves almost to demonstration that the clot has been plugging one ureter; and if, in addition, the clot is of such a size as to support the idea of being a mould of the ureter, the evidence is conclusive as to the source of the hæmorrhage. In renal hæmaturia, minute coagula, casts of the uriniferous tubules, may be discovered in the urinary sediment by the microscope, and may give evidence by their form and shape whence they are derived. In this form of hæmaturia the number of blood corpuscles is no criterion of the quantity of albumin in the urine, whereas in hæmorrhage from the conducting or collecting portions of the urinary tract, provided there is no pus in the urine, the number of red corpuscles or the amount of hæmoglobin may be accepted as the measure of the quantity of albumin contained in the excretion.

(*b*) *The Admixture of other Deposits with the Blood.*—In many cases of hæmaturia the only abnormal constituent in the urine is blood, while in other instances it is mixed with pus, mucus, tuberculous material, portions of tumours, or micro-organisms, the detection of which throws considerable light on the etiology of the malady. The mixture of the blood with deposits of another kind requires only to be mentioned here. The three most common are pus, mucus, and tuberculous débris. If the urine is placed in a conical glass and allowed to stand for a few hours, and the glass is then held up to the light, if pus is present in any great amount the deposits will be found in distinct layers like geological strata; the bottom of the glass is occupied by a yellowish deposit, which may be more or less blood-stained pus, or, in some cases of renal pyuria, the pus may carry down almost all the blood corpuscles, so as to leave an almost quite clear supernatant fluid. When mucus is present, on the other hand, not uncommonly the blood corpuscles may be thrown down first, forming a layer at the bottom of the vessel, and following upon this may be seen a layer of glairy gelatinous material, which may have a reddish tint, or may contain a multitude of minute bloody streaks which intersect it, and penetrate into all parts of this gelatinous layer. In such cases the hæmorrhage is usually due to some lesion in the bladder, and the presence of such an amount of mucus is generally indicative of a more or less acute vesical catarrh. The tuberculous deposit can only be distinguished by microscopic examination, by the cultivation of tuberculous bacilli, or by inoculation experiments. Beyond demonstrating the presence of blood-casts of the uriniferous tubules the microscope may reveal the existence of other elements of consequence in the deposit. For example, the character of the epithelium as derived from the renal parenchyma, from the pelvis, the ureter, or the bladder, may greatly assist one in diagnosis; or, on the other hand, should the urine contain small fragments of tumours, parasites, or bacteria, the hæmorrhage may be explained.

(c) *The Time at which the Blood appears in the Stream.*—In order to ascertain the precise moment at which the blood appears in the stream it is necessary for the surgeon to see the patient urinate, and to observe whether the coloration is equal during the whole continuance of micturition, or is more abundant at the beginning or at the end of the act. The appearance of blood at the beginning of micturition, the remainder of the flow being clear, may be an indication of two distinct conditions—the lesion is either in the prostate close to the neck of the bladder, or a malady or injury is present in the urethra. In some lesions of the prostate the blood may pass into the urethra and accumulate there; so also in lesions of the first portion of the urethra the blood may flow backwards into the bladder and mix with its contents. When the blood originates in the urethra and accumulates there, or when it escapes from the prostate and flows into the urethra, the hæmaturia is limited to the beginning of micturition; but if the quantity of blood is great, and flows backward into the bladder and mixes with its contents, then the whole of the urine becomes more or less coloured. Again, in a lesion at the neck of the bladder giving rise to hæmorrhage, the hæmaturia is not limited to the beginning of micturition, but blood is also observed to escape at the end of the act. This is easily explained. Between the acts of urination the blood accumulates in the urethra or close to its internal orifice, and is expelled with the first few drops of urine only, that following being clear; but again, before complete contraction of the bladder occurs, a fresh hæmorrhage is induced, which shows itself in the urine last ejected. When the hæmorrhage is due to injury or disease of the urethra, the nature of the lesion is generally clearly indicated by the circumstance that the blood is observed to escape quite independently of micturition; sometimes, indeed, blood is seen to flow from the meatus spontaneously. In cases of tumour of the bladder, and also in vesical calculus, the hæmorrhage is most profuse at the end of micturition.

(d) *The Frequency and Duration of the Attacks.*—While nothing absolutely definite can be ascertained by a close observation and study of the frequency and duration of the hæmaturia, still considerable help may be got in this way to aid in a diagnosis. For example, in many cases of renal hæmaturia the blood may suddenly appear and just as suddenly disappear, soon to be followed by a profuse recurrence; such sudden transformations are in some cases accompanied by the expulsion of long worm-shaped clots, and in such instances we may reasonably conclude that the sudden clearing of the urine has been due to the ureter being obstructed. The blood has coagulated within its lumen, and when the clot becomes displaced a fresh, and often apparently very profuse, escape of blood is observed. When hæmaturia comes on without being evidently provoked, we may generally surmise that the lesion giving rise to it is a serious one, although we may not be able to judge its site. In cases of tumour of the bladder the presence of blood is generally very persistent, without intervals, and of long duration, so that the patient may become very anæmic from loss of blood. Again, on the other hand, we meet with cases where hæmaturia more or less profuse has recurred at frequent intervals; but the duration of the attacks has been short, and the intervals between them marked by complete absence of any blood in the urine. Sometimes there may be periods of relief extending over several weeks or even months, and usually any fresh recurrence of bleeding can be explained by excessive exercise or unwonted freedom of movement on the part of the patient. If these conditions are associated with the absence of vesical symptoms, the strong presumption is in favour of the hæmorrhage being renal in origin and against the hypothesis that the blood is flowing from an ulcerated surface; the total duration of the symptoms is also an important guide to the benign or malignant nature of the malady giving rise to it.

(*e*) *The Effects of Movement or Exercise or of Complete Rest in the Course of an Attack.*—When complete rest is taken, hæmaturia due to the presence of stone in the renal pelvis, in the bladder, or in the prostate, is generally more or less relieved; so also in hæmorrhage observed in cases of movable kidney, or in passive hyperæmia of the kidney resulting from pressure on the renal veins. In such cases it is repeatedly observed that the blood is most abundant in the urine at night when the patient has been taking active exercise during the preceding day, while at the same time the pain in the renal region or irritability of the bladder is increased. It must, however, be also borne in mind, in exceptional instances, that in hæmaturia from an abraded surface, as in carcinomatous or tuberculous ulceration, or in senile prostate, the bleeding occasionally may not be increased by exercise; but, as a rule, if the hæmaturia persists in spite of prolonged rest in bed, and especially when the bleeding is more abundant during the night than at other times, carcinomatous, sarcomatous, or tuberculous ulceration is to be looked for.

II. *By collecting the Urine separately from the two Ureters, or by observing Blood escaping from the Orifice of a Ureter.* — The cystoscope may be used both in men and women, and when properly employed the examination subjects the patient to very little risk. The sound is too frequently employed in cases of hæmaturia. It is only when the bleeding is the result of a vesical calculus, or an enlarged prostate, or an hypertrophy of the bladder, that any reliable information can be gained by the employment of the sound; and when the hæmaturia is due to other lesions, much harm may be done by the rough manipulation necessary to examine the bladder with that instrument. The educated use of the cystoscope, being more gentle, is less dangerous, and it is more valuable; but it should always be used with strictly aseptic precautions. In all cases, not only should the cystoscope itself be thoroughly sterilised, but the meatus and surrounding parts should also be carefully washed, and rendered as free from contaminating particles as possible; and after examination has been made the bladder should be carefully washed out with a fresh supply of suitable antiseptic solution. By making careful examination with the cystoscope (*vide* "Cystoscope," vol. ii.) it is usually easy to ascertain whether or not the blood is from the bladder; but in a few instances it is not safe to conclude from the fact of no lesion being observed in the bladder that the hæmaturia is therefore either renal or urethral in its origin. When, however, the blood is seen by the cystoscope flowing from the orifice of a ureter, or more rarely from both ureters, the observation is of the highest importance as indicating the source from which the blood comes.

Catheterisation of the Ureters.—Catheterisation of the ureter in the female has been much simplified by Kelly of Baltimore.[1] The essential feature of his method is that of the introduction of a straight speculum into the empty bladder. The walls of the viscus are slightly separated by the position assumed by the patient, the dilatation being such as to bring the orifices of the ureters into view when reflected light is thrown into the bladder by a forehead mirror. Those who desire to catheterise the ureters must carefully study their direction in the various parts of their course, and must always remember that gentle manipulation is of the highest importance.

Within the last few years two catheterising cystoscopes have been introduced, the one by Nitze, and the other by Casper. In catheterising the ureters it must be borne in mind that rough introduction of the catheter may of itself lead to hæmorrhage, and certainly this is a drawback to its use, especially when the catheterised ureter is the seat of inflammation. In the female the procedure is easily carried out, and the results are tolerably certain; but in the male, even when one succeeds in introducing the catheter into the ureter, it is difficult to say to what degree the results obtained are to be relied upon.

A point of considerable importance in diagnosis is the appearance of the orifice of the ureter as seen by the cystoscope.

III. *By carefully considering all the other Objective and Subjective Phenomena.*—The questions which have been considered up to the present give but a rough sketch; the detail requires to be carefully filled in before the picture is completed. In order to do this the practitioner must consider the most common sources of hæmaturia, and enumerate the various lesions of the kidney, the ureters, the bladder, the prostate, the urethra, and the testicles, which may give rise to the symptom under discussion. It is well always to conform to this rule in going over the etiological factors which have been indicated above.

Placing aside for the present the hæmaturia due to hydatid disease, the renal hæmorrhages of the greatest practical importance, from our present standpoint, are those arising from injury, renal calculus, tumours of the kidney, and tuberculous disease of that organ. Having determined that the hæmorrhage is not from the lower urinary tract, it remains to ascertain which of those four causes is to blame.

When the hæmorrhage is due to *renal calculus* it is usually small in amount, sometimes constantly present, but generally with more or less prolonged intervals, and commonly oft-repeated. In some instances, however, the hæmaturia is very slight, while in other cases it may be the only symptom. The bleeding is not closely related to pain or to the development of other symptoms, but as a rule it is increased by movements of the body. This, however, does not follow immediately, but a short interval may elapse—hours, or even days—between the exertion and the appearance of the blood in the urine. While the hæmaturia of renal calculus is more copious after exercise, rest in bed usually appreciably diminishes it. This peculiarity is most characteristic. The blood, it must be borne in mind, is derived from the renal pelvis and not from the parenchyma; consequently renal blood-casts are not found in the urine, and the quantity of albumen is fully explained by the presence of blood. When the urine is evacuated the blood is thoroughly mixed with it, but not so intimately as when the cause of the hæmaturia is structural disease of the kidney, and if the urine be allowed to stand for a few hours the blood corpuscles are readily precipitated and leave the supernatant urine clear. If free from blood the urine will also be found to be non-albuminous. The presence or absence of pus in the urine will be determined by the circumstance whether or not the calculus has induced inflammatory changes, and the existence of a swelling in the renal region will depend upon the amount of freedom for the escape of urine by the ureter.

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[1] *Twentieth Century Practice of Medicine*, 1895, vol. i. p. 675.

Renal hæmorrhage from *tumour* is generally more profuse and less transient than from calculus, and in not a few cases it is so copious as to cause marked anæmia—a result seldom induced by calculous hæmaturia. It is often developed without any preceding pain. In calculous disease injury or exercise commonly provokes the bleeding, and therefore one finds the hæmorrhage more profuse during the day while the patient is moving about. The bleeding from tumours is, on the contrary, most likely to occur during the night, while the patient is at rest in the recumbent posture. The urinary deposit may assist one in the diagnosis. Concretions composed of oxalate of lime, uric acid, phosphates, or urates may indicate the character of the stone; while by carefully filtering the urine in cases of tumour, portions of the growth may be procured for microscopic examination. The presence of a persistent swelling in the renal region, associated with considerable hæmaturia, is of significance, and may be held as clearly indicating the presence of a neoplasm in the kidney. Exceptions to this rule have, however, been recorded by Ebstein, Hirtz, and Fleming. But while this is so, it must not be forgotten that the presence of a palpable new formation may for a considerable time be preceded by the presence of blood in the urine. Considerable distension of the pelvis may be produced by a calculus obstructing the ureter, and so lead to a swelling in the renal region, or the bulging in the loin may be caused by enlargement of the kidney from tuberculous disease; in neither of these conditions, however, does hæmaturia frequently occur. In the latter the very obstruction which causes an increase in bulk of the kidney prevents the hæmorrhage.

Although much difficulty is often experienced in distinguishing calculous hæmaturia from that caused by tumours, there is still greater care required in the diagnosis between the hæmorrhage of early tuberculous disease and that of renal calculus. The symptoms of the two latter conditions are sometimes identical. This is especially so when the patient fails to show any other evidence of tuberculous disease than that revealed by the renal lesion.

In *tuberculous disease* hæmaturia is frequently absent for long intervals, is seldom so severe as in stone, and is not increased by exercise. In both conditions pus may be mixed with the urine; but while in the latter concretions and gravel may be discovered, in the former careful and repeated search may demonstrate the presence of tuberculous bacilli. In the early stage of tuberculous disease of the kidney the quantity of albumin in the urine is generally in excess of that accounted for by the blood, and in the later stages, when pus appears in considerable quantity, the pus and blood are not so rapidly or so completely precipitated from the urine as in calculous pyelitis. The presence of phthisis pulmonalis, tuberculous disease of bones or joints of the testicle, the prostate, the vesiculæ seminales, the mesenteric glands, the intestine, or of the lower urinary tract, may give a clue to the cause of the hæmaturia.

Hæmatoporphyrinuria.

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See also PIGMENTS.

THE condition known as hæmatoporphyrinuria has attracted considerable attention of recent years. It is characterised by the passage of urine of a dark red colour, like that of port wine, and which, although free from hæmoglobin, contains a considerable amount of that derivative of the blood pigment which is known as hæmatoporphyrin.

This employment of the term, although very convenient, is open to certain objections. In the first place, hæmatoporphyrin is present in minute quantity in normal human urine, and under various morbid conditions the amount present is notably increased, so that it is sometimes possible for a trained eye to detect some of its characteristic absorption bands when a sufficiently deep layer of the urine is examined with the spectroscope. However, under ordinary conditions the quantity present is too small to have any material influence upon the colour of the liquid.

Secondly, in the condition known as hæmatoporphyrinuria there is not merely an exaggeration of the increased hæmatoporphyrin excretion, which is so common a phenomenon in disease, but rather a profound disturbance of pigment metabolism of which this is only one of the evidences. These dark red urines owe only a small part of their abnormal colour to the hæmatoporphyrin which they contain, as Hammarsten was the first to point out, and their tint is mainly due to the presence of other abnormal pigments of which we as yet know very little. Moreover, even the known pigments which they contain are apt to exhibit certain peculiarities in their behaviour to solvents, and in other respects.

The frequent dependence of such hæmatoporphyrinuria upon the administration of sulphonal is no longer open to doubt, and the allied drugs trional and tetronal appear to have a similar action, only in a lesser degree. In the great majority of the recorded cases the urinary change has formed one of a group of toxic symptoms following upon the taking of sulphonal for a longer or shorter period. However, of the many patients who nowadays take this hypnotic, only very few ever exhibit such symptoms, and it is a remarkable fact that with few exceptions those who so suffer are women. The few recorded cases in males have been for the most part of a mild character.

Patients taking sulphonal do not, under ordinary circumstances, excrete unusual amounts of the pigment in question, and those who develop hæmatoporphyrinuria have often taken sulphonal nightly, for weeks or months, with impunity. Sometimes the toxic symptoms only make their appearance after the drug has been discontinued, and in one of Hammarsten's cases the interval was as long as nine days. Sometimes, again, they have followed the administration of very few doses.

Of the associated symptoms, those most frequently observed are vomiting, constipation or diarrhœa, abdominal pain, and paresis or paralysis of limbs. In severe cases the patients pass into a state of collapse, with cyanosis and coldness of the extremities, and death occurs at an early period. In favourable cases, on the other hand, the attendant symptoms quickly subside, and the urine gradually regains its natural colour.

The changes found post-mortem in fatal cases have not been very characteristic. Submucous hæmorrhages have sometimes been present in the stomach and intestines, but are by no means constant. The liver usually shows conspicuous fatty degeneration, and granular or necrotic changes in the epithelium of the renal glomeruli and tubules bear witness to a toxic nephritis.

It appears equally certain that hæmatoporphyrinuria occasionally, but rarely, occurs apart from the administration of sulphonal or allied drugs. In these days of self-medication it is not always easy to exclude their use in a given case, but there are examples on record in which this could be definitely excluded, and some of these occurred before the introduction of sulphonal.

As a rule these cases have run a favourable course, but in two recorded by Ranking and Pardington, one of which proved fatal, other symptoms were present similar to those observed in sulphonal cases.

Neusser met with hæmatoporphyrinuria in association with phthisis pulmonalis in one case, and with pleurisy with effusion in another; MacMunn in a case of exophthalmic goitre; MacCall Anderson saw repeated attacks in association with recurrences of hydroa æstivalis. Nebelthau has recorded its occurrence in a case of congenital syphilis, and Sobernheim in the case of a boy admitted to hospital as suffering from typhoid fever. The only case of this kind which I have had the opportunity of watching was that of a woman admitted to hospital for hæmatemesis, and who was under the care of Dr. Calvert. Although no evidence of the taking of sulphonal could be elicited by careful inquiry, the characters of the urine were very similar to those met with in the sulphonal cases, the dark colour being mainly due to abnormal pigments other than hæmatoporphyrin.

In cases of hæmatoporphyrinuria the colour of the urine usually resembles that of tawny port-wine, but it may be so dark as to approach to black. As a rule it is free from albumin, but by the aid of the centrifuge a few tube-casts may usually be detected. The reaction is usually acid, often strongly so, and the urine may usually be kept for a long time without decomposition. In some sulphonal specimens, which Hopkins and I examined, there was no increased excretion of iron.

Direct spectroscopic examination does not usually yield such satisfactory results as might be expected considering the amount of hæmatoporphyrin present. The other abnormal pigments cause much general absorption of the blue end of the spectrum, and often greatly interfere with the view of the absorption bands. However, the narrow band in red of alkaline hæmatoporphyrin can usually be made out (Fig. 2).

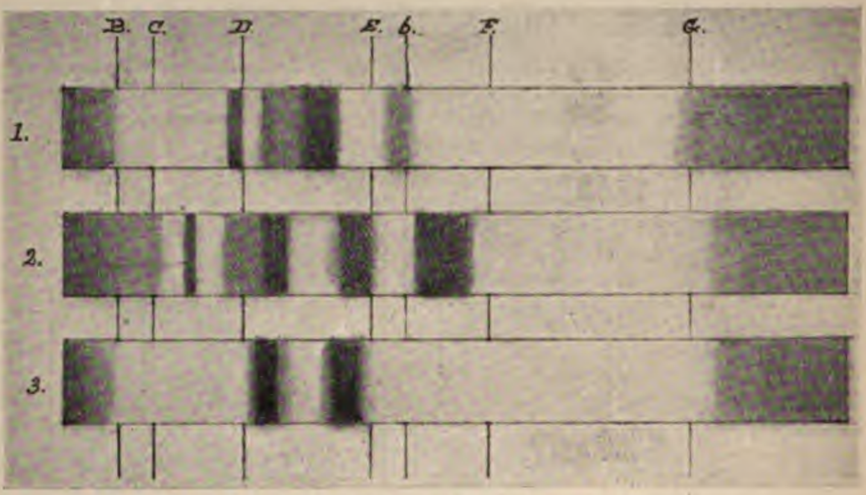
Frequently the bulk and sometimes the whole of the hæmatoporphyrin present is in the form in which it shows what is known as the "metallic" spectrum (Fig. 3), consisting of two dark bands resembling those of oxyhæmoglobin, and this introduces a further difficulty in its detection by direct spectroscopic examination. The addition of hydrochloric acid causes the acid spectrum (Fig. 1) to appear, but this spectrum is never seen, even with strongly acid urine, until a mineral acid is added.

More satisfactory results are usually obtained by extraction with acetic ether after the addition of acetic acid. By this means a solution of hæmatoporphyrin is obtained comparatively free from the associated pigments, but occasionally it refuses to be taken up even by this solvent.

The method of precipitation of the pigment with the earthy phosphates, by adding a 10 per cent solution of sodium hydrate (20 c.c. for each 100 c.c.

of urine), and extraction from the washed precipitate with acid alcohol, although it serves admirably for the detection of hæmatoporphyrin in normal and ordinary morbid urines, does not usually give good results in these cases. Salkowski's method of precipitation with equal volumes of 10 per cent solution of barium chloride and baryta water is certain in its action, but has the disadvantage that the acid alcoholic extract obtained from the precipitate contains the other abnormal pigments. Nebelthau succeeded in precipitating the hæmatoporphyrin by adding glacial acetic acid to the urine (5 c.c. acid to 100 c.c.) and allowing it to stand for 24-40 hours.

Of the processes which are at work in the production of hæmatoporphyrinuria, we know very little as yet. Stokvis found that when sulphonal was administered to rabbits hæmatoporphyrin appeared in their urine. Kast and Weiss were not convinced that the pigment excreted under these circumstances was really of this nature, but Stokvis's observation has recently been confirmed by Otto Neubauer. Stokvis also found submucous hæmorrhages in the stomach of rabbits poisoned with sulphonal, which gave the spectrum of acid hæmatoporphyrin, and he was inclined to attribute the hæmatoporphyrinuria to the action of the gastric juice aided by sulphonal upon the blood so extravasated. However, the records of post-mortem examinations in fatal cases of this condition lend little support to this theory, for although such hæmorrhagic areas have sometimes been found, their presence is by no means constant.



Spectra of Hæmatoporphyrin.—1. Acid; 2. Alkaline; 3. Metallic.

Blood counts made in a number of cases have failed to show any very conspicuous or progressive diminution of red corpuscles in association with the hæmatoporphyrinuria, and the evidence available points rather to a perversion of the ordinary chemical processes for the disposal of effete blood pigment than to excessive hæmolysis as the cause of the phenomenon.

It should be mentioned that in some instances the administration of trional has been followed by the passage of dark urine rich in urobilin, but without any marked excess of hæmatoporphyrin. Urobilin is also usually present in considerable amount in the ordinary dark red urines.

As to treatment little can at present be said. In sulphonal cases the immediate discontinuance of the drug is clearly indicated. Franz Müller attached much value to treatment with alkalies in his case, and a trial should certainly be given to this plan, which receives support from some experiments of Stokvis upon rabbits. Two cases which have come under my observation, in which alkalies were given, ran a favourable course, but neither was originally of a severe character.

LITERATURE.—**Sulphonal cases**: Stokvis. *Nederl. Tijdschr. v. Geneesk.* 1889, p. 413; *Zeitschr. f. klin. Med.* 1895, xxviii. p. 1.—Salkowski. *Zeitschr. f. physiol. Chemie*, 1891, xv. p. 286.—Hammarsten. *Skandinav. Archiv f. Physiol.* 1892, iii. p. 319.—Müller, Franz. *Wiener klin. Wochenschr.* 1894, vii. p. 252.—Garrod and Hopkins. *Journal of Pathol. and Bacteriol.* 1896, iii. p. 434.—Schultz (trional). *Deutsche med. Wochenschr.* 1894, xx. p. 152.—Herbin (trional). *Ibid.* p. 343.—Oswald. *Glasgow Med. Journal*, 1895, xliii. p. 1.—Kast and Weiss. *Berliner klin. Wochenschr.* 1896, xxxiii. p. 621.—Neubauer, Otto. *Archiv f. exper. Path. und Pharmakol.* 1900, xliii. p. 456.—Rolleston (urobilinuria). *Clin. Soc. Trans.* 1897, xxx. 125.—Schulte. *Deutsches Archiv f. klin. Med.* 1897, lviii. p. 313. Only a small number of the recorded sulphonal cases are included in the above list. **Cases not due to sulphonal or allied drugs**: Neusser. *Wiener Sitzungsberichte*, Abth. iii. 1881, lxxxiv. p. 536.—Ranking and Pardington. *Lancet*, 1890, ii. 607.—Sobernheim. *Deutsche med. Wochenschr.* 1892, xviii. p. 566.—M'Call Anderson. *Scottish Med. and Surg. Journal*, 1897, i. p. 97.—MacMunn. *Journ. Physiol.* 1890, xi.; *Proc. Physiol. Soc.* p. 13. —Nebelthau. *Zeitschr. f. physiol. Chemie*, 1899, xxvii. p. 324. References to the general literature of hæmatoporphyrin in urine will be given in the article on "Pigments."

Hæmoglobinuria.

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The term hæmoglobinuria is used to designate the passage of urine containing free oxy- or methæmoglobin in solution, or in the form of granular débris, as distinguished from hæmaturia, the passage of urine mixed with blood. It is the outward manifestation of hæmoglobinæmia, by which is meant the presence of free hæmoglobin in the blood-plasma.

Ponfick has shown that if a solution of hæmoglobin, or laked blood, be injected into the circulation of an animal, until a certain proportion is reached the metabolic processes are able to dispose of the introduced pigment, but that if this proportion be exceeded the excess of hæmoglobin is excreted by the kidneys.

A similar result follows transfusion of the blood of an animal of a different genus, under which circumstances the foreign red corpuscles undergo disintegration, and the blood pigment which they contained appears in the urine. The transfusion of lamb's blood into human patients, which was at one time recommended as a remedial measure, was usually followed by hæmoglobinuria, but this is not the case when transfusion is performed from one human being to another, unless the corpuscles have been previously broken up.

There can be no doubt that hæmolysis is constantly in progress in the body, and that in certain diseases, such as pernicious anæmia, its normal limits are greatly exceeded, and we must suppose that under such conditions the metabolic processes which dispose of the effete blood pigment are not over-taxed, or that the hæmolysis takes place elsewhere than in the general circulation. In the conditions which are attended by hæmoglobinuria, on the other hand, the destructive process goes on with great rapidity, and apparently in the general circulation.

Jaundice of greater or less degree frequently accompanies hæmoglobinuria, and it may be looked upon as another manifestation of hæmoglobinæmia. This fact was held to supply one of the strongest arguments for the existence of a hæmatogenous variety of jaundice, due to the change of blood pigment into bile pigment without the intervention of the liver. More recent researches, however, tend to show that even in these cases the jaundice is obstructive, and results from changes in the consistence of the bile, partly due to the formation of an excess of bile pigment, and partly to an increased secretion of mucus, as a result of irritation of the biliary passages.

Toxic Hæmoglobinuria.—A great variety of toxic substances possess the hæmolytic power requisite for the production of hæmoglobinuria. And of these the most important, because it is so extensively used as a drug, is potassium chlorate, which appears to share with other chlorates the additional property of converting oxy- into methæmoglobin in the circulation, and even within the red corpuscles. Arseniuretted hydrogen is also a hæmolytic poison, and Eitner and others have recorded cases in which hæmoglobinuria has resulted from the inhalation of hydrogen containing this gas as an impurity. Amongst other such poisons may be mentioned toluylene-diamine, the effects of which upon animals have been carefully studied by Stadelmann, William Hunter, and others; nitro-benzol, hydrochloric and sulphuric acids, carbolic acid, pyrogallol, naphthol, and sulphuretted hydrogen.

Even quinine must be mentioned among the substances which are credited with giving rise to hæmoglobinuria, especially in malarial cases (*see* article "Blackwater Fever" in vol. i.).

Bostroem has described some cases of poisoning by a mushroom (*Morchella esculenta*) in which hæmoglobinuria was a prominent symptom.

Hæmoglobinuria in Fevers.—Hæmoglobinuria may also occur in the course of various specific fevers and in septic diseases. It is seldom observed, and as a rule in severe cases. Thus Immermann met with it in a relapse of typhoid fever, and Cnopf and Huebner have described its occurrence in scarlatina. Hayem and Robin have seen hæmoglobinuria as a complication of acute rheumatism, and the present writer has seen a very temporary hæmoglobinuria in a case of this nature, and also in one of lobar pneumonia. In both instances it was of very short duration; the urine was pink and quite limpid, containing hæmoglobin, but no blood corpuscles. In malarial fevers hæmoglobinuria sometimes occurs, and it is a prominent symptom of the so-called "blackwater fever," which owes its name to this circumstance.

Hæmoglobinuria in the Lower Animals.—Hæmoglobinuria is a prominent symptom of an epidemic disease which occurs in cattle, and in which Babes found a micrococcus-like organism in the blood, and especially in the kidneys.

Horses also are liable to a peculiar affection which attacks heavy draught animals, on resuming work after a few days' rest and good feeding. Its onset appears to be favoured by cold. Hæmoglobinuria is a prominent symptom, and it is attended by an acute form of myositis. The attacks may be prevented by reducing the food and giving the animals some exercise during the resting period.

In this connection it is interesting to note that Chvostek found that hæmolysis was more easily produced in horses than in other animals.

Infantile Hæmoglobinuria.—Winkel has described a remarkable epidemic which occurred among new-born infants in the lying-in institution in

Dresden, in which hæmoglobinuria was a prominent symptom. In all, twenty-three infants were attacked in quick succession, and of these no less than nineteen died. The onset was usually on or about the fourth day of life, and the average duration of the illness was thirty-two hours. The other leading symptoms observed were cyanosis, some degree of jaundice, which was often very slight, coldness of the extremities, and unconsciousness. There was no febrile disturbance in any case, but rather a rapid fall of temperature.

The blood was found to have a treacly consistence and a dark brown colour. There was a marked leucocytosis, and much granular detritus was present.

The post-mortem appearances recalled those met with in cases of potassium chlorate poisoning, and included deep pigmentation of the renal cortex, hyperæmia of the pancreas, enlargement of the spleen and mesenteric glands, dilatation of the stomach, and ecchymoses in the stomach and intestines, with swelling of Peyer's patches.

Numerous micrococci were found in the blood, kidneys, and intestine.

Winkel considered that umbilical infection might be excluded — no medicinal or dietary cause could be traced, and it is noteworthy that all but five of the affected infants were breast-fed, and the mothers showed no impairment of health. Sporadic cases, apparently of the same nature, have since been recorded by Sandner and Baginsky.

Paroxysmal Hæmoglobinuria.—Of the conditions which give rise to the symptom under discussion, the most remarkable, and in many respects the most interesting, is the affection known as paroxysmal hæmoglobinuria, which appears to be a disease *sui generis.*

The earliest clear description of this malady, as one characterised by the intermittent passage of urine deeply coloured by blood pigment and containing hæmoglobin débris, but free from red blood corpuscles, is contained in a paper by George Harley in the *Medico-Chirurgical Transactions* for 1865, which is immediately followed by a record of some further cases by W. H. Dickinson. It is true that the disease does not appear to have wholly escaped the notice of earlier observers, and reference may be made to the description of a case of intermittent albuminuria and chromaturia by Dressler, published in 1854. In a paper by Alexander Marcet, published in 1823, reference is made to a case, apparently of this nature, in which the vaso-motor phenomena were so well marked that it may be regarded as an example of Raynaud's disease.

Paroxysmal hæmoglobinuria is a decidedly rare disease. It commences usually in adult life, before middle age, but it is also met with in children. A very great majority of all the sufferers are males, the recorded cases in females being very few in number.

Malaria and syphilis have been regarded as important predisposing causes, but the more recent investigations suggest that malarial antecedents play a less important part than the earlier observers supposed; whereas it has been shown that a history of syphilis is to be obtained in a large proportion of cases, and children who suffer from the disease have usually suffered from congenital syphilis also.

The initial attack is not infrequently ascribed to some unusually severe exposure, and beyond question cold is by far the most potent exciting cause of the individual paroxysms. In some cases the effect of cold appears to be aggravated by exertion, and there is a remarkable group of cases in which muscular exertion of a particular kind appears to be the sole provoking cause of attacks. Since, however, it is doubtful whether these cases are rightly included in the present category, it will be best to discuss them separately.

The influence of cold is best shown by the fact that in susceptible subjects an attack may be started by a cold bath, and even by the immersion of portions of the body, such as the hands or feet, in ice-cold water, for a longer or shorter period. However, individual patients differ remarkably in their susceptibility to such influences, and a degree and duration of exposure which will in one case inevitably provoke an attack, will in another have no such result.

In addition to hæmoglobinuria a variety of more general symptoms accompany the attack, such as pains in various parts of the trunk and limbs, rigors, febrile disturbance, and, in the later stages, sweating. Headache is often present and may be severe, and gaping or yawning is a common early symptom. Nausea, anorexia, vomiting, and a peculiar sensation of difficulty of swallowing, are also described or observed; and in some cases an urticarial rash appears during the attack.

Among the most conspicuous phenomena are vaso-motor disturbances, such as coldness of the extremities, and "deadness" of the fingers and toes, which are the seat of tingling and numbness, and appear white and bloodless, or even assume a purple tint. These changes are attended by a rise of blood-pressure due to contraction of the peripheral vessels. In a word, a condition is developed which is indistinguishable from the slighter forms of Raynaud's disease, and even the more severe Raynaud phenomena are sometimes met with in association with hæmoglobinuria. It would, perhaps, be more correct to say that hæmoglobinuria is sometimes seen in Raynaud's disease, for whereas vaso-motor disturbances of minor degree are usually observed in paroxysmal hæmoglobinuria, it is only in a few cases of typical Raynaud's disease that hæmoglobinuria occurs.

However, the association is sufficiently striking to warrant the supposition that there is some intimate relationship between the two conditions, rather than that, as Bristowe and Copeman suggested, their association is merely due to the fact that exposure to cold is the common exciting cause of both maladies. Both diseases are decidedly rare, and the probability of their accidental association in the same individual is consequently small.

Recent observations suggest that the vaso-motor disturbances play an important part in the causation of paroxysmal hæmoglobinuria, a second factor being an abnormal condition of the blood, and it seems possible that in cases of Raynaud's disease without hæmoglobinuria this second factor is wanting.

Tenderness in the region of the liver and spleen have occasionally been observed, and even palpable enlargement of those organs.

The general symptoms above mentioned vary in individual cases, and only a selection of them is usually present. However, in any given case the symptoms tend to be fairly constant, each successive paroxysm resembling those which have gone before. Thus one patient stated that he always experienced numbness of all his fingers and of his great toes, whilst in another case the paræsthesia was always most marked in the ring and little fingers, and especially in those of the left hand. Again, the distribution of the pains in the trunk and limbs tends to repeat itself. In one case the attack will always be ushered in by a pain down the back, in another by aching in the loins and thighs.

The number and intensity of the symptoms present are also apt to vary with the severity of the attack, which, again, is usually in proportion to the degree and duration of the antecedent exposure to cold.

Unless some permanent renal affection be present the urine shows nothing abnormal in the intervals between the attacks. The onset of hæmoglobinuria usually occurs an hour or so after the exposure, and it may be preceded by a simple albuminuria. In some instances the urine has contained an excess of urobilin after the attack, but I have never observed this. Possibly it only occurs when the attack is accompanied by jaundice.

The characteristic urine is usually acid and often strongly so, but in some recorded cases it has been constantly alkaline. The specific gravity is as a rule lowered (1010-1015).

Its colour varies from ruby red to that of porter, and in thick layers it may appear almost black. The blood pigment may be present in the form of oxy- or methæmoglobin, and most frequently the spectroscope shows the bands of both these pigments.

The presence of methæmoglobin is evidenced by its characteristic band in the red, and that of oxyhæmoglobin by the intensity of its bands near D and E, which far exceeds that of the corresponding bands of the spectrum of methæmoglobin.

Copeman found that, in his case, when the urine was drawn off by catheter at short intervals it showed the bands of oxyhæmoglobin only, whereas when it remained longer in the bladder it contained methæmoglobin. In some cases examined by the present writer, the band in red of methæmoglobin was clearly visible immediately after the urine was passed, even when the bladder had been emptied not long previously, but the specimens were not drawn off by the catheter. Copeman further believes that, on standing, a portion of the excreted hæmoglobin undergoes a further change to acid hæmatin, and this is one of the many points connected with this disease which calls for further investigation. The presence of methæmoglobin is no special feature of the urine of paroxysmal hæmoglobinuria, nor, indeed, of hæmoglobinuria in general as distinguished from hæmaturia. Hoppe Seyler stated that blood pigment is always passed in the urine in the form of methæmoglobin, and that it changes to oxyhæmoglobin on standing. That such a change takes place in ordinary smoky urines it is easy to convince one's self, but the rate of change varies widely in different specimens.

The most striking microscopical feature of the urine is the absence of red blood corpuscles. It is true that a few are sometimes seen in the sediment, but they bear no proportion to the colour, and their presence is not improbably due to temporary changes in the kidney resulting from the unwonted calls upon them. Such renal changes have been observed in animals after transfusion of foreign blood.

The urine deposits a copious dark brown sediment, which consists of granular hæmoglobin débris, and sometimes of urates mixed with such débris. Casts of renal tubules composed of brown granules are usually present in large numbers, as they also are in the urine of the various forms of toxic hæmoglobinuria and of transfusion cases.

Crystals of calcium oxalate are very frequently seen in such urines, but as yet there is no evidence that their presence has any special pathological significance.

The free passage, through kidneys ordinarily proteid-tight, of such large quantities of proteid material is a matter of considerable interest, which may be compared with the excretion in the urine of other foreign proteids, such as egg albumin, after injection into the circulation of an animal. Some recent researches of Brodie suggest, however, that the physical properties of hæmoglobin may aid in its elimination, as it is found to pass more readily through a perfused kidney or a Martin's filter than either serum albumin or globulin.

Serum albumin is also usually present in small amount in the dark urine of the paroxysm, and some albuminuria may persist for a time after the hæmoglobinuria has passed away. Again, in some cases slighter exposures are followed by albuminuria, and more severe ones by hæmoglobinuria; and Ralfe suggested that the transitory or paroxysmal albuminuria of apparently healthy individuals is really a minor manifestation of the disease under discussion—its "petit mal," as Bristowe happily styled it.

The low specific gravity of the urine passed during the paroxysm suggests a diminished excretion of the heavier urinary constituents, due to blocking of the renal tubules with hæmoglobin débris, and in some forms of toxic hæmoglobinuria the renal secretion has been completely suppressed. Boas was inclined to attribute many of the attendant symptoms to this cause, and the effect of the attack upon nitrogenous excretion is a point which calls for investigation.

Blood counts made before and during the attacks show a marked fall in the numbers of the red corpuscles. Copeman has observed falls varying from 129,000 to 824,000 per cubic millimetre. The destruction is most active at the very commencement of the attack, before the hæmoglobin appears in the urine.

Hayem observed, and Chvostek has confirmed the observation, that blood drawn during the paroxysms clots with unusual rapidity, and that the clot quickly liquefies again. This peculiarity is not met with in blood taken during the free intervals.

The presence of free hæmoglobin in solution in the serum during the attack has been repeatedly observed, both in serum from which blood-clot has separated and in that obtained from blisters. Copeman has occasionally observed the formation of crystals of hæmoglobin in serum which has been allowed to stand.

One of the most obvious microscopic changes is the absence of rouleaux formation by the red corpuscles. Poikilocytosis is sometimes noticed, and particles of free pigment are seen in the plasma. The blood platelets are usually increased in number. As a rule there is no leucocytosis.

Ehrlich, Boas, and other investigators have laid special stress upon the presence of decolorised red corpuscles (*Blutshatten*) as a characteristic feature.

During the exposure which excites the attack there is, as Bristowe and Copeman pointed out, an initial fall of temperature, which is quickly followed by a decided rise, during which 101° or even 103° F. may be reached. Shortly after the maximum has been reached a rapid fall commences, warmth returns to the extremities, and the urine regains its normal character, although slight albuminuria may outlast the hæmoglobinuria for a time. The paroxysms commonly last some five or six hours, but their duration is to some extent proportional to their severity. Occasionally febrile disturbance is absent, and the temperature may even remain subnormal throughout.

The patient is left somewhat anæmic, especially if several attacks have followed each other in quick succession, but the destroyed blood corpuscles are quickly replaced. As in other varieties of hæmoglobinuria, some degree of jaundice may follow the attack, but this is usually slight in character, and is by no means constantly present.

As might be expected, the attacks are most frequent in winter, and many patients enjoy complete immunity from them during the summer months. Even in winter the disease remains in abeyance as long as the patient is protected from cold, as, for example, during a stay in hospital.

Seeing that in the intervals between the paroxysms the patient enjoys practically unimpaired health, except during the short period required for the replacement of the destroyed blood, and since the attacks themselves are not attended by danger, it is not remarkable that the disease has practically no fatal tendency, and may continue for many years without serious detriment to health.

Pathology.—It will be seen from what has gone before that the main features of the attack of paroxysmal hæmoglobinuria are—1st, the active hæmolysis which occurs on exposure to cold; and, 2nd, the vaso-motor phenomena by which it is accompanied.

It is probable that many of the minor symptoms are direct results of hæmoglobinæmia. If one refers back to the records of cases of transfusion of lamb's blood into human beings, one is struck by the similarity of the symptoms observed with those of the disease under consideration. Rigors and febrile disturbance were prominent effects, and among other symptoms mentioned, in addition to hæmoglobinuria, were pains in the back and lumbar regions, vomiting, dyspnœa, sweating, and urticarial eruptions. This urticaria followed quickly upon the transfusion, and therein differed from that which is so common a result of the modern serum treatments. It is further of interest to note that rigors, fever, and sweating were observed by Eitner in his cases of poisoning by arseniuretted hydrogen.

On the other hand, rigors and febrile disturbance may follow transfusion from man to man, and were also observed in some cases in which transfusion of milk was resorted to.

One of the main points to which investigation has been directed is the obvious fact, that in the subjects of paroxysmal hæmoglobinuria the red corpuscles show an abnormal tendency to undergo rapid destruction under influences which in ordinary individuals are insufficient to bring about such a result. This susceptibility of the red corpuscles is not a congenital condition, but is acquired at some period of the patient's life, often apparently as a result of an exceptional exposure, and when once acquired it tends to persist.

The researches of Ehrlich, Chvostek, and others, have clearly demonstrated that in the sufferers from paroxysmal hæmoglobinuria the corpuscles are not unduly liable to disintegration, outside the body, under the influence of cold, and Ehrlich was consequently inclined to attribute their intravascular disintegration, under the influence of exposure, to the secretion of a hæmolytic ferment by the walls of the vessels in the part cooled.

Chvostek has shown, on the other hand, and Mannaberg and Donath have recently confirmed his observation, that the red corpuscles of these patients are abnormally sensitive to mechanical influences, such as shaking the blood in a tube, or the jarring of a centrifuge which does not run smoothly. Mannaberg and Donath have further shown that they are broken up with unusual readiness under the influence of carbon dioxide.

Chvostek also found that in repeating the classical experiment of Ehrlich, viz. placing an elastic ligature upon the patient's finger and immersing it in ice-cold water, the cooling was not necessary for the production of local hæmoglobinæmia, but that the simple arrest of the circulation leads to this result. Following Murri, he looks upon the vascular spasm as playing an equally important part with the abnormal blood condition, the presence of both factors being necessary for the production of the attack. Such a theory will explain very well the relationship of Raynaud's disease to paroxysmal hæmoglobinuria, the cases in which the former is present without hæmoglobinuria being those in which the second factor, viz. the undue liability of hæmolysis, is absent. In support of his view that the vasomotor disturbances, aided by the resultant presence of carbon dioxide in excess, brings about the destruction of the abnormally frail blood corpuscles, Chvostek adduces the fact that he was able to cut short the attacks by the administration of amyl nitrite at the earliest stage. Moreover, he found that in horses, whose blood corpuscles appear to be less stable than those of other animals, he was able to bring about a degree of hæmoglobinæmia by the induction of vascular spasm by electrical stimulation of the upper portion of the cervical cord.

Against this must be set the failure of Mannaberg and Donath to cut short the attacks by amyl nitrite in their case, and these authors object to Chvostek's theory that it is not at all evident in what way vascular spasm can place the blood under conditions at all comparable to those present in the shaking experiments, nor do they think that the facts can be more satisfactorily explained by a theory based upon their results with carbon dioxide, venosity of the blood being held wholly responsible for the hæmolysis.

The experiment with the ligature of a finger shows that local hæmolysis may take place in such cases in the peripheral circulation, and the seat of the blood destruction during the paroxysm has been variously located, several observers regarding the renal circulation as the most likely locality. Against this latter view is the presence of free hæmoglobin in the serum generally, in spite of its abundant excretion in the urine. Chvostek's observations led him to the opinion that only a small proportion of the red corpuscles are unduly subject to disintegration under mechanical influences, and if this be so, it points to the tendency to break up being inherent in the corpuscles themselves rather than dependent upon any abnormality of the medium in which they are suspended.

Seeing that so many poisons possess hæmolytic powers, and are capable of causing hæmoglobinuria, the possibility suggests itself that such a toxic substance may be present in the blood of these patients in amounts too small for the direct manifestation of its hæmolytic action under ordinary conditions, but which, nevertheless, is able to bring about blood destruction when aided by the other conditions which excite an attack.

However, such an hypothesis receives no countenance from some remarkable observations of Mannaberg and Donath, who found that whereas the serum of blood obtained from a patient during an interval, and immediately centrifugalised, caused no hæmolysis when added to healthy blood, the serum of an attack, which was slightly coloured by hæmoglobin, became more deeply coloured, and showed the absorption bands more clearly, after it had been added to healthy blood and again separated. A similar hæmolytic action was exercised by serum in which hæmolysis had been mechanically brought about.

The fact that hæmoglobinuria is an occasional symptom of malarial fever, and the part which ague appears to play as a predisposing cause in some cases of paroxysmal hæmoglobinuria, suggest a community of origin, and there are features in the attacks which lend colour to this hypothesis; but, on the other hand, the paroxysms of hæmoglobinuria are in no sense periodic, and their recurrence is strictly dependent upon exposure to the exciting cause. The more recent observers are disposed to agree with Murri in regarding syphilis as a far more important predisposing cause, and a history of malaria is comparatively seldom to be obtained in cases met with in this country.

The scanty number of recorded post-mortem examinations of the subjects of paroxysmal hæmoglobinuria throw practically no fresh light upon the problem of its pathology, the changes met with being similar to those seen in other conditions accompanied by hæmolysis and hæmoglobinuria. In some cases interstitial changes have been observed in the kidneys, which possibly have been results of the unusual work which these organs are at intervals called upon to perform. That this is not entirely without effect upon the kidneys is suggested by the presence of small quantities of serum albumin, and occasionally of a few red corpuscles in the urine during the attacks.

It will be evident, then, that in spite of the large amount of careful investigation which has been directed upon its study the pathology of paroxysmal hæmoglobinuria is still to a large extent obscure, and that no theory of its causation which has as yet been put forward affords a full and satisfactory explanation of the observed phenomena.

Treatment.—Removal of the patient from the conditions which provoke the attacks is one of the prime desiderata in the treatment of the disease, and, when circumstances permit, residence in a climate in which no extremes are encountered, or at least a winter sojourn in a warm climate, are clearly indicated, and may serve to procure complete immunity. In the majority of instances measures directed to the cure of the disease, and to enabling the patient to encounter exposure to cold with impunity, have less satisfactory results. A complete cure has occasionally been brought about by antisyphilitic treatment in the hands of Murri and others, and good effects have sometimes followed the administration of quinine in full doses.

In the treatment of the individual paroxysms warmth is indicated, and a warm bed and warm drinks add greatly to the comfort of the patient, even if they do not curtail the attack. The administration of nitrite of amyl, which Chvostek found effectual in cutting short the paroxysm, should receive a trial, but as the hæmolysis occurs at so early a stage, it is obvious that, if it is to produce any effect, it must be given at the very first onset of the symptoms. Nitro-glycerine might also prove of service as a prophylactic. Copeman recommends the avoidance of alcoholic drinks on account of the temporary dilatation of the superficial capillaries which they produce, which may bring about a slight lowering of the body temperature.

Hæmoglobinuria from Exertion.—The cases in which hæmoglobinuria is induced by muscular exertion are few in number, but of great interest. As they differ in certain not unimportant respects from the ordinary cases of paroxysmal hæmoglobinuria, they are best considered provisionally as constituting a separate group. Examples have been recorded by Fleischer, Kast, Robin, Köster, Lee Dickinson, and others. In some instances at any rate the attacks are frequently repeated, and may be set up even by a very moderate exertion, in others a single attack follows a severe muscular strain, such as is involved in running a three-mile race.

It has been demonstrated in some cases that such exposure to cold as might be expected to induce a paroxysm in an ordinary case of paroxysmal hæmoglobinuria was not capable of producing such an effect, even when an attack followed a moderate walk.

Perhaps the most curious feature of these cases is the apparent powerlessness of other forms of exertion than those involving leg-work to induce the attacks. Marching, walking, running, and in one of Lee Dickinson's cases a hard game of lawn tennis, are the recorded causes, and a patient who develops hæmoglobinuria after a walk may be able to exert himself at wood-chopping with impunity. Another noteworthy feature is that the attendant symptoms which are usually so prominent in paroxysmal hæmoglobinuria are usually absent, and the abnormal coloration of the urine may alone attract attention. Köster's patient, however, complained of coldness of the extremities, chills, and slight headache, but showed no febrile disturbance. This observation is opposed to the view which attributes many of the attendant symptoms to hæmoglobinæmia, but the difference may be merely one of degree, for in some of the exertion cases the amount of hæmolysis, as evidenced by the condition of the urine, seems to have been comparatively slight. In a case described by Bastianelli albuminuria without hæmoglobinuria was observed after exertion on some occasions. Köster's patient, who had acquired syphilis four years previously, recovered completely under antisyphilitic treatment, and in Robin's case recovery followed treatment by rest, tonics, and a restricted diet. In this latter instance, before recovery was complete, exertion which would formerly have induced hæmoglobinuria caused only slight albuminuria.

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Hæmophilia.

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Hæmophilia, or the congenital hæmorrhagic diathesis, is a congenital hereditary disease, characterised by a liability to spontaneous hæmorrhages, to excessive bleedings from trivial causes, and to joint affections. The morbid tendency persists throughout the life of the affected individual, who is termed a "bleeder."

Ætiology.—Heredity and sex are important factors in the production of the disease, yet its actual cause is unknown. Thus the disease occurs in males, females being very rarely affected. Transmission does not as a rule take place directly from father to son, but almost always through seemingly healthy members of the female line, who are often exceedingly fertile, to the male descendants. A somewhat similar mode of transmission of disease is observed in pseudo-hypertrophic paralysis, Friedreich's ataxia, and occasionally in diabetes insipidus and colour blindness. Apart from heredity, there is no obvious cause for hæmophilia, which is met with in all the human races, in various countries and in all ranks of society. Consanguinity of the parents, and a history of gout in the ancestors, are probably merely accidental circumstances.

Morbid Anatomy.—Post-mortem there is anæmia of the tissues and organs, and in many instances also ecchymoses, blood extravasations into the organs, or effusions into the body cavities. Other changes, such as fatty degeneration of organs, thinness of the arterial coats, and abnormally narrow vessels, are not constant, nor are they causal factors. The joints may present morbid appearances from hæmorrhages into their cavities, but in more chronic cases there is thickening of the synovial membrane, with fibrillation, and degeneration of the articular cartilage, the changes in short resembling those seen in rheumatoid arthritis. There is no proof that the blood differs from the normal as regards its chemical constitution. When a bleeding first starts the microscopic appearance of the blood is quite normal, and coagulation is normal also. But if the blood be examined after a severe hæmorrhage, there will necessarily be signs of anæmia, and the blood "resembles water in which fresh meat has been washed and scarcely stains linen" (Legg). Coagulation, too, is then very deficient, or does not occur at all. The essential nature of the disease, in short, is unknown, though generally held to consist either in some abnormality of the blood-vessels, or in an abnormal state of the blood whereby clotting is retarded or altogether in abeyance.

Symptoms are classified under three headings: (1) Spontaneous or traumatic external hæmorrhages; (2) Interstitial hæmorrhages; (3) Joint affections.

1. In a small percentage of cases the disease manifests itself soon after birth by severe umbilical hæmorrhage. As a rule, however, the child is about a year old before any signs appear, the subjects of hæmophilia being usually quite normal as regards their general appearance, physiognomy, nutrition, etc. The earliest sign is often a severe external hæmorrhage after some trivial wound, and as the boy grows older he suffers frequently from these uncontrollable bleedings after various forms of traumatism, such as slight cuts, tooth extraction, circumcision, opening of abscesses, etc. The hæmorrhage is usually capillary, and is characterised by its severity and the great difficulty with which it is checked, blood oozing away slowly but steadily, it may be for days or weeks in succession. Or the bleeding may be spontaneous, and is then more often from the mucous and serous surfaces. In early life these bleedings often occur as severe epistaxes, less commonly, as bleedings from the gums, and after the beginning of the second decade there is a greater tendency to intestinal, urethral, renal, gastric, or pulmonary hæmorrhages, than in infancy. Effusion of blood into the peritoneal cavity is occasionally seen.

2. The interstitial hæmorrhages are either spontaneous or occur after traumatism. In the former case there are petechiæ or ecchymoses in the

skin during infancy or later life, and hæmorrhages in the mucous membranes, tissues, and organs. The ecchymoses in no way differ in appearance from similar conditions found in persons with an acquired hæmorrhagic diathesis. Traumatic ecchymoses are common, and allied to them are hæmatomata, which may attain the size of a man's head.

3. Joint affections do not usually arise till the fourth or fifth year, and the larger joints are the more commonly affected. Spontaneous or traumatic hæmorrhage occurs into the joint cavity (hæmarthrosis), and there is an inflammatory infiltration of the periarticular tissues. Thus the joint appears swollen, is painful, especially on movement, and the overlying skin is often somewhat discoloured. The onset of the swelling is often sudden, and accompanied by a rise of the temperature to 103°-104° F. The swelling usually disappears in the course of a few weeks, but tends to recur. In consequence of repeated intra-articular hæmorrhage changes occur in the joint structures, giving rise to appearances closely resembling rheumatoid arthritis, and in later stages the ends of the bones are much thickened, and the joint is more or less anchylosed, flexed, and useless.

Diagnosis is easy in well-marked cases. The milder varieties may present more difficulty; important points to remember are that the disease is hereditary and mainly affects males, that the first signs appear as a rule before the tenth year, and that the disease lasts throughout life. It may be mistaken for—(i.) *The hæmorrhagic diseases of the new-born.* Idiopathic umbilical hæmorrhage affects both sexes equally, and leaves no subsequent liability to excessive hæmorrhages. Melæna neonatorum usually occurs within a few days of birth, is due to gastric or intestinal ulceration, and is often fatal within a week. Some forms of congenital syphilis and the hæmoglobinuria of Winkel's disease are readily differentiated, the latter being accompanied by cyanosis, jaundice, and somnolence, and being probably an infective disease. (ii.) *Scurvy*, the various forms of *purpura* and the hæmorrhages of gout, leucocythemia, etc., are as a rule easily distinguished. (iii.) Lastly, the joint affections must not be confounded with *rheumatism, gout, rheumatoid arthritis*, or *tubercular disease* of the joints.

Prognosis.—The younger the patient the graver is the prognosis; but with care adult life is often attained, and the hæmorrhagic tendency possibly diminishes after middle life.

Treatment.—If there is known to be an hereditary tendency to the disease, the children, and especially the boys, should be carefully reared, and all operative measures avoided, with the exception of vaccination, which is hardly ever attended with special risk. Older children and adults have usually been warned by their relatives of their morbid tendency, but some bleeders are peculiarly reticent in admitting that they are sufferers; hence, if hæmophilia be suspected, mere denial thereof should not entirely allay our suspicions. If traumatic hæmorrhage has occurred, general surgical measures must be employed to arrest the bleeding; operative interference, however, causes hæmorrhage as uncontrollable as that from the initial lesion. Compression, combined, if necessary, with plugging of the wound, is the most efficient treatment, especially if complete rest be observed at the same time. Styptics are useless, but freezing with ice or ethyl chloride has sometimes acted well. Internal medicaments, *e.g.* chloride of calcium, ergot, or acetate of lead, may also be tried, but are of more use for checking bleedings from mucous surfaces. Substances, such as nucleic acid, yeast, thymus, and other preparations containing nuclein, are recommended on account of the fact that they produce leucocytosis. Transfusion may be resorted to if other measures fail. Treatment which appears beneficial in one case is often useless in another, in which the bleeding continues unchecked, only ceasing when syncope ensues, or even causing the death of the patient. Joint swellings are best treated by means of compression and immobilisation. After a bleeding the patient usually remains somewhat anæmic for some months and the administration of iron, cod-liver oil, and other tonics, with attention to the hygienic surroundings, is advisable.

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Hæmoptysis.

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DEFINITION.—The expectoration of blood from the lungs or lower air-passages (larynx, trachea, and bronchi).

PATHOGENESIS.—The older writers were in the habit of using the terms *Bronchorrhagia* and *Pneumorrhagia* as signifying in the one case effusion from the bronchial mucous membrane, and in the other escape of blood from the pulmonary vessels proper. Laennec was of opinion that slight hæmoptyses were bronchial in origin, while all grave hæmorrhages were due to pulmonary apoplexy, the result of vascular tension in the lungs themselves. In this he was in error. Pulmonary apoplexy is due to embolism, and is a comparatively rare cause of hæmoptysis.

All pathological evidence goes to prove that in the vast majority of cases of hæmoptysis—both slight and severe—the pulmonary and not the bronchial vessels are to be regarded as the source of the hæmorrhage. This is only what we would theoretically expect when we consider how badly supported the pulmonary vessels are as compared with those of the bronchial mucous membrane. The hæmorrhage itself is due either to escape of blood by *diapedesis*, the result of active or passive hyperæmia of the pulmonary, and to a much less extent of the bronchial capillaries; or, on the other hand, to *rupture of a blood-vessel*, generally a branch of the pulmonary artery, the result of some pathological condition (*e.g.* ulceration or aneurysm) of the vascular wall.

ETIOLOGY.—A. *Diseases of the Lungs.—Pulmonary Tuberculosis.*—This, especially in its more chronic forms, is *by far the most important cause of pulmonary hæmorrhage.*

Sir Thomas Watson says: "If a person spits up blood who has received no injury to the chest, in whom the uterine functions are healthy, and who has no disease of the heart, the odds that there are tubercles in the lungs of that person are fearfully high."

Hæmoptysis occurs in about 60 per cent of all cases of pulmonary tuberculosis; the Inpatient Brompton Report gives 62 per cent; Dr. Williams (1000 private cases) gives 57 per cent; Dr. Wilson Fox, 54 per cent. Of 200 successive cases admitted by the writer into Brompton Hospital, hæmoptysis occurred in 70 per cent. It is extremely rare as a symptom of acute miliary tuberculosis. Hæmoptysis occurring in pulmonary tuberculosis is due to one of the following causes:—

(1) *Hyperæmia and Rupture of Capillaries.*—Occurs mainly in the early stages of tuberculosis. Such hæmorrhages are usually very slight, and are indicative of congestive and inflammatory states of the pulmonary tissue. Dr. Kingston Fowler regards capillary hæmorrhage as probably the cause of the streaks and small blood-clots which appear from time to time in the sputum in the course of chronic pulmonary tuberculosis.

(2) *Tubercular Infiltration with subsequent Ulceration and Perforation of a Branch of the Pulmonary Artery.*—Tubercle bacilli have been demonstrated by Koch, Weigert, and Percy Kidd in the thickened and infiltrated vessel walls in connection with commencing caseation. The ulcerative process occasionally leads to perforation of an artery of some magnitude, and in this way may induce very profuse hæmorrhage. This is probably the cause of the severe hæmoptyses which occasionally occur in the earlier stages of pulmonary tuberculosis, particularly when associated with rapid caseation. Fortunately such a result is comparatively rare, owing to the fact that closure of the vessel by thrombosis and endarteritis obliterans generally takes place. In the more rapid forms of tuberculosis, the vascular wall becomes softened and infiltrated before the thrombotic process is complete, and rupture takes place, the vessel being no longer able to withstand the force of the blood-pressure within.

(3) *Rupture of an Aneurysm of a Branch of the Pulmonary Artery.*—This is by far the most common cause of profuse hæmoptysis, and occurs most frequently in the stage of excavation. In 30 out of 35 consecutive fatal cases occurring at Brompton Hospital, Dr. Percy Kidd traced the source of the bleeding to ruptured pulmonary aneurysms, while in 3 out of the remaining 5 unruptured aneurysms were discovered. These aneurysms, which are usually the size of a large pea or hazel nut, but which may reach the size of a walnut or even larger, are most commonly found where an artery passes along the wall of a cavity. The vessel wall, which may be softened by tubercular infiltration, bulges on the side next the cavity where it is not supported by lung tissue. Very rarely the vessel may be seen to bulge into the lumen of a bronchus. Occasionally an aneurysm has been found in a caseous patch which is undergoing softening. The aneurysms are usually round and sacculated, but may be fusiform or irregular. Very occasionally they contain laminated blood-clot. They are commonly single, but not infrequently multiple; as many as thirty have been found of various sizes.

Besides tuberculosis, hæmoptysis, especially in slight amount, may occur in practically all diseases of the lungs. In *bronchiectasis*, hæmoptysis frequently occurs in the later ulcerative stages. Dr. Fowler observed it in 14 out of 35 cases. The hæmorrhage is usually small in quantity, but is sometimes profuse, as much as half a pint of blood may be expectorated. In *carcinoma of the lung* the sputum is frequently blood-stained, and not uncommonly resembles red currant jelly or prune juice, and may rarely contain cancer-cells. In *hydatids* hæmoptysis occurs in the majority of cases. There is usually simply a staining of the sputum, but a severe hæmorrhage may occur prior to rupture of a cyst. Dr. Percy Kidd records one case of fatal hæmoptysis from this cause. In *cirrhosis of the lung*, which may be due to dust inhalation, syphilis, pleuritic extension, or other cause, the slighter degrees of hæmoptysis are not uncommon. According to Dr. Bastian, it occurs in half of all cases. That form of cirrhosis described by Sir Andrew Clark as non-tubercular fibroid phthisis, in which hæmoptysis is not an infrequent symptom, is considered by most modern authorities to be identical with an extremely chronic form of tuberculosis affecting chiefly the supporting framework of the lung, with subsequent fibroid transformation of the tuberculous areas. Hæmoptysis is a rare symptom of *emphysema*, but does occasionally occur and may even prove fatal. It is usually, however, small in amount. It is matter for surprise that it does not occur more frequently considering the frequent association of atheroma of the pulmonary artery with emphysema. In *lobar pneumonia* we have the typical blood-stained, viscid, rusty sputum. Hæmoptysis also occurs in *gangrene, pulmonary abscess,* and *actinomycosis.* Streaked expectoration may be observed in cases of *bronchitis,* especially after a paroxysm of coughing.

B. *Diseases of the Heart.*—Hæmoptysis is specially frequent in mitral disease, and more particularly in mitral stenosis. It occurs also in aortic regurgitation. The hæmorrhage is due to rupture of pulmonary capillaries from backward pressure, and is liable to occur after exertion. Large hæmoptysis is not infrequently met with in mitral stenosis. The writer has seen several cases where there have been recurring hæmorrhages exceeding ¼-½ pint in quantity.

C. *Aortic Aneurysm.*—Frequently terminates by rupture into trachea, bronchi, or lung. It seems hardly necessary to state that such hæmoptysis is in the nature of the case rapidly fatal, though there is not uncommonly a pink staining of the sputum for some days or longer due to "weeping" of the aneurysm.

D. *Embolism or Thrombosis of the Pulmonary Artery* causes *pulmonary apoplexy* or hæmorrhagic infarction. The embolus may come from the heart, or may be transmitted from a thrombosed peripheral vein, as in phlegmasia alba dolens. If a fatal termination does not rapidly ensue, there may be fairly profuse hæmoptysis for some considerable time.

E. *Constitutional and Morbid Conditions of the Blood—e.g.* Leucocythæmia, purpura hæmorrhagica, scorbutus, hæmophilia, malignant infective fevers. In regard to hæmophilia, some authorities consider that the hæmorrhage is due, not to an alteration in the blood, but to an hereditary structural imperfection of the inner coats of the vessels. A *senile* hæmoptysis has been described occurring in persons over fifty years of age. These may occur and recur in arthritic subjects (Sir Andrew Clark) without any serious disease being present.

F. *Traumatic.*—Blood is expectorated after injuries to thorax and lungs, *e.g.* bullet or sword wounds, or fractured ribs; wounds of larynx and trachea, *e.g.* cut throat; also after operations on larynx, trachea, or lungs, *e.g.* tracheotomy, paracentesis thoracis (by suction), evacuation of empyema, or pulmonary abscess.

G. *Inflammations and Ulcerations of Larynx, Trachea, and Bronchi—e.g.* Tuberculosis, syphilis, carcinoma, foreign bodies, extension of œsophageal epithelioma.

H. *Vicarious.*—Hæmoptysis occurs in rare instances at the menstrual epochs in cases of suppressed menstruation, or at the menopause, or as the result of plethora. It is stated that periodical hæmoptyses have been observed to follow the removal of both ovaries. Sir Thomas Watson mentions a remarkable case of a woman who menstruated through her lungs at each monthly period for forty-two years (from 16 to 58 years of age).

I. *Endemic Hæmoptysis*—due to the presence in the lungs of the Distomum pulmonale—has been described by Drs. Ringer and Manson. Is endemic in *Japan, Korea,* and *Formosa.* The disease is characterised by chronic cough with expectoration of a peculiar rusty-brown pneumonic-like sputum. The patient is liable to irregular attacks of hæmoptysis, which may be profuse. In the rusty-brown sputum there are abundant dark-brown, thick-shelled operculated ova. They are oval, smooth, and double outlined, measuring from 80 to 100 μ in length by 40 to 60 μ in breadth. In the course of a month or six weeks a ciliated embryo is developed in each ovum, which, on escaping at once, begins to swim about in the water. The life-history is probably continued in some fresh-water animal, through which it finds its way back to man. Small areas, rather larger than a filbert—the so-called "burrows"—are found scattered throughout the organ, particularly towards the periphery. These "burrows" contain a number of "tunnels" filled with the characteristic rusty-coloured material, and each containing one or more small distoma. The septa between the tunnels may break down, producing a considerable cavity.

The distomum pulmonale v. Ringeri v. Westermanni is reddish brown in colour, oval in form, and so thick and fleshy that it is almost circular in transverse section. It measures 8 to 10 mm. by 4 to 6 mm., and is covered by minute spines.

Exciting Causes of Hæmoptysis.—Very often there is no exciting cause, but an attack is sometimes determined by muscular exertion, a paroxysm of coughing, mental agitation, menstruation, straining at stool, alcoholism, or extremes of heat and cold.

Pathological Appearances.—In the case of a fatal hæmoptysis the mucous membrane of the air-passages is usually blood-stained. Blood is found in the trachea, bronchi, and lungs. There may be a thick cylindrical clot extending from the trachea down to the smaller bronchi. The blood is as a rule most abundant in the vicinity of the ruptured vessel, though it is often present in parts quite remote, and not infrequently a large amount of blood-clot may be found in the opposite lung. This flooding of the sound lung with blood is commonly the cause of death in fatal cases by asphyxia. Blood may even be found in the upper lobe of the opposite lung, showing that the inspiratory efforts have been powerful enough to force the blood up against the action of gravity. As regards the heart, it presents the appearances characteristic of death from asphyxia or syncope. In cases which recover, the effused blood is usually completely absorbed; but Dr. Reginald Thomson has shown that in a number of cases the relics of blood are to be found in the presence of hard, fibrinous, pigmented nodules. In cases of fatal hæmoptysis due to pulmonary embolism we find large hæmorrhagic infarctions. In cases which recover, these alter into hard, wedge-shaped fibrinous masses.

Putrefactive changes very rarely occur in the effused blood except in those cases where the hæmorrhage is the result of inhalation of blood from the parts above the larynx (*e.g.* epistaxis and mouth operations), when it is due to the presence of the accompanying putrefactive organisms.[1]

Symptomatology.—The onset of an hæmoptysis may be sudden or preceded by one or two days of staining. Occasionally there is a preceding sense of tightness across the chest. There is usually in tubercular cases a previous history of cough and expectoration, though in about 3 per cent of cases hæmoptysis is actually the first symptom. It is to be noted that the onset is not as a rule associated with exertion or strain, as one might suppose. The patient simply experiences a tickling sensation about the fauces, has a saltish taste in the mouth, and suddenly discovers that his mouth is full of bright blood. The amount expectorated varies in quantity from a mere streak or staining of the sputum to one, two, or more pints of blood. In early phthisis the bleeding is usually small in amount, but is liable to recur. Profuse hæmoptysis generally occurs in the later stages. In Dr. Pollock's return of 341 cases of large hæmoptysis 12 per cent occurred in the first stage, 41 per cent in the second, and 46 per cent in the third. The blood is generally bright red and frothy, but when very profuse is often dark and clotted, especially if it has gradually accumulated in pulmonary cavities or bronchial tubes. When the flow is not excessive the blood is often mixed with sputum. Blood-casts of the smaller bronchi are occasionally expectorated. Usually the expectoration remains blood-stained for some days after, or there are brownish black specks in the sputum.

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[1] *Phthisis ab Hæmoptæ.*—Hæmoptysis was such a common and striking feature of consumption that the older writers, from Hippocrates downwards, erroneously regarded phthisis itself as directly due to infective and putrefactive changes in the effused blood. In later times Niemeyer himself reverted to this old view, basing his opinion on the fact that sometimes hæmoptysis is really the first symptom, and also on the fact that pyrexia not uncommonly follows an hæmoptysis. Since, however, the discovery by Koch of the pathogenic tubercle bacillus it has been discovered that this after-pyrexia described by Niemeyer is in reality due to infective tuberculous broncho-pneumonia of other portions of the lungs. The term is therefore a misnomer.

In the case of a profuse hæmoptysis the patient is generally greatly alarmed—the face is pallid and bedewed with sweat; there is faintness, feeble pulse, and coldness of extremities. The blood is brought up with a frequent short cough, and the patient may continue to bring it up for some hours. In severe cases blood may pour out at the mouth and nose, and may rapidly fill a fairly large porringer (or small basin). Some of the blood is often swallowed, and is either vomited or passed per rectum. Owing to the collapsed condition of the patient the temperature becomes subnormal. When the shock has passed off the temperature rises generally within twelve hours to normal or to its original height before the hæmorrhage occurred. In a few instances a high temperature may be observed for some time afterwards, due probably to the formation of foci of infective broncho-pneumonia, the result of insufflation of the lungs with blood charged with micro-organisms from the cavities and softened areas. Dr. Williams regards this after-pyrexia as of ill omen. It may be some time before the patient recovers from the mental depression and from the ever-present dread of its recurrence. The majority of all cases do tend to recur. The term *hæmorrhagic phthisis* has been applied to those cases where hæmorrhage is a constant and frequent phenomenon. This frequent recurrence may be explained by the supposition that the rent in the wall of the artery or aneurysm has only undergone partial repair.

It may be here noted that it is quite possible for a fatal hæmorrhage to occur into a large cavity without any blood being expectorated at all.

As regards the *examination of the chest* it is very important to remember that for some time after a severe hæmoptysis the patient should be disturbed as little as possible. He should not be asked to take a deep breath or to cough or speak, and the chest should on no account be percussed. The lungs may be auscultated during tranquil respiration without risk. In addition to the signs of the accompanying lung disease one can often hear fine moist crepitant râles over the area of effusion, and, according to Laennec, these are frequently audible in the vicinity of (just above) the roots of the lungs. These may persist for some days, or even weeks.

DIFFERENTIAL DIAGNOSIS.—A. *Differentiation of the various causal Conditions that lead to Hæmoptysis.*—They are as a rule readily distinguished by their associated symptoms and clinical features. Owing to its extremely frequent occurrence in connection with phthisis the presumption in the absence of heart disease is always in favour of tuberculosis, even although no physical signs of pulmonary disease can be detected. On the other hand, if a presystolic murmur is heard at the cardiac apex we can generally dismiss all idea of phthisis, as mitral stenosis and pulmonary tuberculosis very rarely coexist. The following points may also be noted: —The sputum in acute pneumonia has the well-known rusty colour: in gangrene it frequently resembles prune juice; in carcinoma it has been likened to red currant jelly; in pulmonary œdema it consists of a sero-sanguineous fluid; in brown induration of the lung, and in a pulmonary apoplexy of some standing, it may be of a dark brown colour, owing to the presence of hæmatoidin crystals.

B. *Diagnosis from other Hæmorrhages.*—(1) *Hæmatemesis.*—In hæmatemesis there is usually a history of dyspepsia, and there is often accompanying pain in the epigastrium. The blood is acid in reaction from admixture with gastric juice; it is dark in colour, and more or less altered by the gastric juice. It is preceded by nausea, and comes up with vomiting, and may be mixed with food. The blood is brought up all at once, and not in successive mouthfuls; faintness often precedes the hæmorrhage; subsequent coffee-ground vomit and tarry motions. In hæmoptysis, on the other hand, there is often an antecedent history of cough or other pulmonary symptoms. The blood comes up with coughing. It is alkaline in reaction, is bright red and frothy, and mixed with mucus. There are often air-bubbles in the coagulum; there are occasionally blood moulds of the smaller bronchi. It is preceded often by a tickling sensation in the throat; is generally followed for some days by stained sputum (this is probably the most important diagnostic point); the hæmoptysis is generally repeated; microscopic examination may reveal tubercle bacilli, elastic fibres, or pulmonary epithelium. (In primary hæmoptysis the expectorated blood should always be stained for bacilli.)

The following points must be noted in order to guard against possible fallacies:—The act of vomiting may excite coughing, and *vice versa;* there may be no cough at the first appearance of hæmoptysis; blood from the lungs may be swallowed, producing secondary hæmatemesis and melæna; and hæmatemesis may occur from rupture of varicose veins at the lower end of œsophagus as a symptom of hepatic cirrhosis.

(2) *Epistaxis.*—In nasal hæmorrhage blood may pass back into the pharynx and be expectorated. Anterior and posterior rhinoscopy may be necessary to decide whether the hæmorrhage arises in the nose or posterior nasal space. Hæmoptysis has been known to occur in connection with undue vascularity of the inferior turbinated bones without epistaxis.

(3) *Hæmorrhage from Throat or back of Tongue.*—Hæmoptysis to any extent from this cause is very rare. Streaks of blood are occasionally observed in the expectoration accompanying some minor diseases of the pharynx. Sometimes the presence of blood in the mouth on rising from sleep has been observed in varix of the tongue, mouth, and pharynx. In this connection it may be noted that real hæmoptysis in childhood is rare, even in cases of pulmonary tuberculosis. When a young child does spit up blood it is generally owing to the violence of the coughing, as in the paroxysms of whooping-cough, and often comes from the throat, nose, or gums, and occasionally from an ulcer of the frenum linguæ.

(4) *Spurious Hæmoptysis and Malingering.*—Neurotic anæmic girls often speak of finding blood on their pillows on waking, or of their mouths being filled with blood in the morning. They acquire the habit of sucking their cheek or gums. The latter are seen to be pale, spongy, and exuding blood. The sputum in these cases is generally, according to Dr. Wagner, of a pale red colour, not so bright as in ordinary hæmoptysis, and on settling sometimes presents a reddish brown sediment. The blood is mixed with saliva, but contains no cylindrical or ciliated epithelium. The blood only appears in the morning, and there are no physical signs in the lungs.

Malingerers may produce blood in the same way, or may actually add blood derived from another source. The late Professor Hughes Bennett discovered a case of malingering by examination of the blood with the microscope. The corpuscles were oval instead of round, thus proving that they did not come from a mammalian source at all.

Prognosis.—Hæmoptysis is very rarely fatal. In the large proportion of cases there is a tendency to spontaneous arrest of the hæmorrhage. Dr. Samuel West states that the mortality from hæmoptysis in phthisis is less than 3 per cent. Sometimes an hæmoptysis occurs without warning, continues for a few days, and then disappears, leaving no traces behind, and no physical signs develop. In all probability, however, there has really been in these cases a tubercular focus, which has cicatrised and healed up. Such foci at one or other apex are frequently found post-mortem. Of 4000 cases of hæmoptysis observed at Brompton Hospital the quantity in 69 per cent was under ℥ss. Still profuse and repeated hæmoptysis must always be regarded as a grave condition. The large majority of fatal cases occur in the stage of excavation, and are due to the rupture of a pulmonary aneurysm. Fatal hæmoptysis is more common in males than in females, and is rare in the case of children and old people, though the writer has seen in the Royal Chest Hospital a fatal termination in the case of a child under three years of age. Dr. Reginald Thomson says that fatal hæmoptysis is more common in the summer months—June and July. Death is due, in the majority of cases, to asphyxia from regurgitation of blood into the bronchial tubes, more particularly of the sound lung. The patient is practically drowned in his own blood. In a few cases death is due to syncope.

As regards the influence of hæmoptysis on the progress of pulmonary tuberculosis opinions vary somewhat. The laity very often regard spitting of blood in quantity as "the beginning of the end." There is certainly no clinical evidence in support of such a view. Dr. Pollock, in his *Elements of Prognosis in Phthisis*, says hæmoptysis, even if profuse, may have no appreciable effect on the subsequent health of the individual. In not a few cases the effects seem actually beneficial. It is easy to understand that this is the case in hæmoptysis due to chronic pulmonary congestion by acting as a safety-valve. May it not occasionally act in a similar way in the acute pulmonary hyperæmias of pneumonia and early tuberculosis? Certainly one frequently sees cases of recurring hæmoptysis with practically no chest symptoms and little if any deterioration of the general health. Dr. Williams, after careful observation, expresses the opinion that the influence of hæmoptysis in the first stage is nil, whereas hæmoptysis occurring in the second and third stages curtails the average duration of the disease.

It must also be remembered that a certain proportion of cases of chronic phthisis may assume a subacute progressive character after an hæmoptysis has occurred. This, as already explained, is probably due to the onset of secondary infective broncho-pneumonia.

Treatment.—The indications are twofold:—A. *Promote thrombosis*, which is the natural process of repair, by lowering the blood-pressure in the pulmonary artery. In this way we arrest the bleeding. B. *Prevent as far as possible the regurgitation of blood* into the neighbouring bronchi, and more particularly into the bronchus leading to the opposite lung; and *promote its expulsion therefrom in the event of its occurrence.* In this way we generally minimise the immediate danger of fatal asphyxia, and also prevent the later occurrence of infective broncho-pneumonia.

A. Thrombosis is promoted by the following means:—(1) *Rest*—Fulfils all therapeutic indications, and is much the most important of the cardiac sedatives. Nothing more is required in mild cases. The syncopal condition of the patient also acts as an excellent cardiac depressant. Rest ought to be as absolute as possible—general, local (cardiac and respiratory), and mental. The patient is confined to bed in a cool, airy room, is kept quiet and undisturbed, is lightly clad, in severe cases is forbidden to speak above a whisper, is only asked essential questions; the cough is checked by sucking pieces of ice or by a sedative linctus. Dr. Mitchell Bruce lays stress on the mental aspect of the question, and makes a point of reassuring the patient by a few encouraging words. He is requested to lie as still as possible. In the European sanatoria *ligature of the extremities* is frequently carried out with the view of diminishing the influx of blood into the lungs, and so checking the hæmorrhage. A special apparatus is employed, but the same effect can be produced by a tight bandaging of the upper arms and thighs.

(2) *Opium*—Is by far the most important drug in the treatment of hæmoptysis. It acts as a cardiac and respiratory sedative and depressant, checks the cough, allays the general restlessness, soothes the mental distress and agitation, and promotes sleep. The best method of administration is by a *subcutaneous injection of morphia* (gr. $\frac{1}{4}$-gr. $\frac{1}{3}$), which may have to be repeated once or even twice. Some physicians recommend a combined injection of morphia (gr. $\frac{1}{4}$) and atropine (gr. $\frac{1}{100}$).

(3) *Low Diet.*—Nothing is given for some hours except small pieces of ice to suck at considerable intervals, so as to prevent flatulence with its accompanying restlessness. The sucking of ice relieves the thirst as well as the cough, and likewise calms the patient's anxiety by giving him the feeling that something definite is being done for his relief. After a little time iced milk is given in tablespoonful doses and gradually increased in quantity. Everything must be cold. Cold milk, cold meat jelly or essence, with a little bread and butter, is usually sufficient for the first few days, along with, it may be, a little pounded meat and milk pudding. Alcoholic stimulants must be strictly avoided, except in the rare instances where there is danger of syncope. The pulse must be carefully watched.

(4) *Saline Purgatives.*—These lower the blood-pressure by acting as derivatives. Dr. Kingston Fowler recommends one drachm of sulphate of magnesia with 20 grains of sulphate of soda given every four hours.

(5) *Ice-bag to Chest*—Is now little used. Has probably no effect on the ruptured vessels, but if applied over the præcordial region may have some effect in lessening the cardiac excitability.

(6) *Other Medicinal Remedies.*—(*a*) *Astringents.*—Though used extensively by the older school of practitioners, their value is exceedingly doubtful. Ergot, which is undoubtedly a valuable drug in uterine hæmorrhage, has the greatest reputation, while hazeline, gallic acid, and turpentine are also employed. Sir R. Douglas Powell speaks well of acetate of lead in combination with dilute sulphuric acid. Hydrastis Canadensis is used by some American physicians. It is not easy to understand what effect such astringents can possibly have on an ulcerated vessel or ruptured aneurysm. Indeed, Bradford and Dean have shown that ergot and hazeline actually raise the blood-pressure in the pulmonary circulation. Clinical experience is also against the use of these remedies. During the writer's term of office as house physician at Brompton Hospital he was never once called upon to administer ergot or any other astringent. They are better withheld altogether, as they derange digestion and induce constipation. (*b*) *Aconite* has been recommended by Dr. Andrew on the ground that it lowers the blood-pressure in the pulmonary circulation (Bradford and Dean).

B. The prevention of the regurgitation of blood and its subsequent expulsion in the event of its occurrence are secured by the following means:—

(1) *Position of the Patient.*—This is of the utmost importance. The patient, on the occurrence of a severe hæmoptysis, should be immediately turned and made to lie on the affected or most affected side. It is the corresponding lung that is the source of the hæmorrhage. If this is done, then any regurgitation of blood that may occur will, owing to gravity, take place from the trachea into the bronchus of the affected lung, where it will do little harm, as that lung is largely functionless, especially in the stage of excavation, when profuse hæmoptysis is so common. On the other hand, if, on the incidence of the hæmoptysis, the patient happens to be lying on his sound side, the blood will regurgitate from the trachea into the bronchus leading down to the healthy functioning lung, and breathing becomes almost impossible. If the patient be lying on his back the blood will regurgitate into both lungs. In the writer's opinion, flooding of the sound lung with regurgitated blood is the most common cause of fatal asphyxia, a result which would often be avoided were this rule always attended to. The writer, while house physician at Brompton Hospital,[1] was in the habit of writing the letters R. or L., as the case might be, on the bed cards of all the cases of phthisis to indicate the affected or most affected side. The charge nurses had instructions, in the event of a hæmoptysis, to immediately turn the patient on the affected side. In spite of the fact that there were numerous cases of profuse hæmoptysis, not a single fatal termination occurred during his term of office. The patient's friends should be told of the importance of position.

(2) *Morphia,* by checking the cough, soothing the restlessness, and calming the mental distress, does much to diminish the frequent inspiratory efforts which conduce to regurgitation.

(3) *Artificial Respiration.*—If there is threatened asphyxia from flooding of the lungs with regurgitated blood, it is advisable at once to have recourse to some form of artificial respiration, with the patient lying on the affected side. Such a case occurred at Brompton Hospital, where, by the use of Sylvester's method of artificial respiration, the writer succeeded in getting most of the blood ejected, with consequent speedy recovery, and this too after the patient's condition had been pronounced hopeless by the physician in charge who happened to be present when the hæmoptysis occurred.

(4) *Stimulating Expectorants.*—After a day or two it is often advisable to give small doses of ipecacuanha, squills, or ammonium carbonate, in order to assist in the expulsion of the small relics of blood from the minuter bronchi. There is thus less likelihood of a subsequent infective broncho-pneumonia.

After-treatment and Prophylaxis.—Diet should be sparing, and alcoholic stimulants avoided for some weeks. The patient may be allowed to get up after the sputum has been free from staining for a few days. Should take occasional laxatives and carefully avoid all excessive exertion. Ferruginous remedies and tonics may be necessary to counteract the anæmia and associated debility. Primary hæmoptysis is a warning of incipient lung disease, and therefore a change of climate is indicated as soon as the patient can bear the journey. Traube spoke highly of the value of respiratory exercises as a preventive measure in cases of chronic recurring hæmoptysis associated with congestion of the respiratory organs.

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[1] Brompton Hospital is the largest consumption hospital in Great Britain. There is provision for 350 patients, of which 80 per cent are cases of pulmonary tuberculosis.

LITERATURE.—*Pulmonary Consumption* (WILLIAMS); *Pulmonary Hæmorrhage* (REGINALD THOMSON); *Prognosis in Consumption* (POLLOCK); *Diseases of the Lungs* (WILSON FOX); *Diseases of the Lungs* (FOWLER and GODLEE); *Tropical Diseases* (MANSON); *Brompton Hospital Medical Reports*; *Hæmoptysis* (Dr. RASMUSSEN), Hospitals-Tedende, Copenhagen, 1868.

Hæmorrhage.

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See also ARTERIES, NEW-BORN CHILD, PREGNANCY.

HÆMORRHAGE is always present when a blood-vessel is wounded, and the loss of blood may be so trivial as to give rise to no definite constitutional effect, or so severe in case of wound of one of the largest arteries as to prove rapidly fatal. The blood may escape externally through an open wound, or may become extravasated subcutaneously, or into one of the great cavities of the body. A patient may die from hæmorrhage without one drop of blood being shed external to the body, *e.g.* intraperitoneal hæmorrhage. The main considerations in estimating the danger of a severe hæmorrhage are the quantity of blood lost by the individual and the rapidity with which the hæmorrhage occurs. Of all surgical emergencies there is none more urgent, and none which makes greater demand upon the judgment and presence of mind of the surgeon than a rapid and severe hæmorrhage, and upon its proper treatment the life of the patient entirely depends.

It is customary to describe four varieties of hæmorrhage, classified according to the source of the hæmorrhage, viz. arterial, venous, capillary, and parenchymatous. In addition to the above we distinguish, according to the time of the occurrence of the bleeding, (*a*) primary; (*b*) reactionary, intermediary or consecutive; and (*c*) secondary hæmorrhage.

1. Arterial Hæmorrhage.—In this variety the blood is bright red (oxygenated), and escapes in jets from the wounded artery synchronously with the beats of the heart. It is to be borne in mind, however, that in patients who are deeply anæsthetised or suffering from partial asphyxia the blood escaping from a wounded artery may be dark-coloured.

2. Venous hæmorrhage is characterised by the dark colour of the blood and by its flowing in a continuous stream, due to the absence in the veins of the impulse of the heart-beat. In general the walls of the vein collapse.

3. Capillary hæmorrhage is characterised by the oozing of blood from several points of the wounded surface. The amount of blood lost at each bleeding point is but small, and it is only rarely when this variety of hæmorrhage persists for several hours that the amount of blood lost is sufficiently considerable to place life in danger. Owing to the fact that the capillaries in the mucous membranes are larger and more abundant than those of the skin, hæmorrhages from the former are apt to be more troublesome than from cutaneous wounds.

4. Parenchymatous hæmorrhage is chiefly characterised by its occurrence in parts or organs which present structural peculiarities, for example in erectile tissue, where little arteries open directly into veins without the intermediation of capillaries, as in the erectile tissue of the genitalia and in the spleen. It is also to be observed in parts whose normal vascular condition is altered, particularly by thrombosis and malignant infiltration.

A. Primary hæmorrhage occurs immediately upon receipt of the injury to the vessel. It may, of course, present the characteristics of any of the above-described anatomical varieties of hæmorrhage.

B. Reactionary, intermediary, or consecutive hæmorrhage occurs after the establishment of the period of reaction, *i.e.* usually within the first twenty-four hours after the receipt of an injury or after a surgical operation. It is due generally either to the slipping of a ligature or to the increasing blood-pressure forcing out of the injured vessels the coagula which plug their divided ends.

C. Secondary hæmorrhage occurs after the expiration of at least twenty-four hours from the time of the injury. It is most frequently observed from the sixth to the fifteenth day. It usually occurs in a suppurating wound, and is caused by ulceration of the walls of the vessel, the result of a septic arteritis; very rarely is it due in an aseptic wound to the too rapid absorption of a catgut ligature or to incomplete repair in the injured artery. Secondary hæmorrhage, in olden days a frequent and very serious occurrence, has now, thanks to surgical cleanliness, become a rare wound complication.

CONSTITUTIONAL SYMPTOMS OF HÆMORRHAGE.—A patient suffering from severe hæmorrhage presents the following appearance. He is pallid, cold, and yet beads of perspiration abound, the lips are blanched, but show a faint lividity, the pulse feeble, quick, and small, and perceptible only perhaps in the large arteries. The breathing is irregular and quickened, the alæ nasi dilate, and complaint is made of shortness of breath, the temperature is below normal, convulsive movements, marked restlessness, and vomiting may occur. There are generally abnormal sensations of sight and hearing, as flashes of light, a sound as of rushing water, or buzzing in the ears, and in extreme cases syncope, unconsciousness, and death may supervene.

Whilst the quantity of blood lost and the rapidity with which the hæmorrhage occurs are, as above stated, the main considerations, other factors have to be taken into account; thus aged persons, and very young children, stand the loss of blood badly, but they differ markedly in their recuperative power—children recovering in cases not rapidly fatal quickly and thoroughly, aged people much more slowly, and indeed rarely completely.

Women at the period of parturition recover from hæmorrhages which at any other time would almost certainly prove fatal.

Hæmorrhagic Fever.—After a serious hæmorrhage the patient frequently presents the condition known as hæmorrhagic fever, in which are found acceleration and sometimes irregularity of the pulse-rate, elevation of temperature, extreme thirst, diminution of the quantity of urine passed per diem, and low muttering quiet delirium. This condition, formerly held to be due to septic absorption, is now generally believed to be in great part due to insufficient supply of blood to the great nerve-centres.

SPONTANEOUS ARREST OF HÆMORRHAGE.—Fortunately in the great majority of cases natural processes stay the blood flow before the hæmorrhage proves fatal. If an artery be divided completely, it, owing to its elasticity, retracts, and its muscular coat contracting narrows or even closes the open wound in the vessel. In this way the loss of blood is lessened or arrested, and coagulation at the seat of injury is favoured. In small arteries muscular contraction may suffice of itself to completely arrest the hæmorrhage. Cardiac syncope not infrequently, by diminishing the pressure of the blood in the artery, acts as a potent factor in arresting hæmorrhage. Coagulation of the blood is also aided by the exposure of the blood to atmospheric influences, and to the curling in towards the lumen of the vessel of its middle and internal coats. All the above factors aid in bringing about the formation of a blood-clot, which lies inside the sheath of the vessel, and is applied to the end of the divided artery, constituting the "temporary external coagulum." A second blood coagulum now forms within the lumen of the severed vessel. This, which is known as the "internal coagulum," frequently extends upwards as far as the next large branch of the vessel. The external coagulum becomes in course of time entirely absorbed, whilst as the result of the subsequent organisation of the internal coagulum the artery becomes completely obliterated at the seat of injury. In lacerated wounds of arteries the middle and internal coats curl upwards and inwards into the lumen of the vessel, and the external coat and sheath become twisted over the opening. Hence the fact that lacerated wounds of vessels scarcely bleed appreciably. In wounds of veins retraction and contraction are not so perceptible as in the case of divided arteries, but the collapse of the wall of the vein greatly aids the formation of a coagulum.

DIAGNOSIS OF HÆMORRHAGE.—When the blood escapes externally no difficulty in making a diagnosis of hæmorrhage can occur. Very different, however, is the diagnosis of concealed hæmorrhage, as when the blood finds its way into one of the great cavities of the body, the peritoneal cavity for example. In such cases, though most of the constitutional symptoms of severe hæmorrhage be present, they frequently are only by the employment of the greatest care able to be diagnosticated from those of shock. In the great majority of cases the physical signs enable a correct diagnosis to be made.

TREATMENT OF HÆMORRHAGE.—Constitutional treatment is called for after a severe hæmorrhage (the bleeding having been, if possible, first arrested). Owing to the general weak state of the patient energetic treatment is often called for. The head should be placed as low as possible to prevent anæmia of the brain, and the patient be kept absolutely at rest, the windows of the room should be opened to ensure a free supply of fresh air, the patient should be warmly covered with blankets. The arms and the legs may be elevated, or better, encircled in Esmarch's rubber bandages, in order that blood may be driven out of them into the brain and the thoracic viscera. Large quantities of warm fluids may be given to the patient to drink, or better, a copious enema of water, 1 to 1½ pints, may be injected into the large bowel, and will generally be retained. Heart stimulants, such as strychnine, carbonate of ammonia, or better, hypodermic injections of ether or brandy, are very beneficial. There is, however, a danger of producing over-stimulation, so that these remedies need to be judiciously administered. In very extreme anæmia from hæmorrhage, when the above-mentioned means fail, there still remains one remedy which has often saved life in the most desperate cases, and which should never be omitted, viz. the transfusion of a sterilised ·07 per cent of sodium chloride (see "Transfusion").

It may be well here to point out that the cause of death from hæmorrhage is dependent solely upon mechanical conditions. Death occurs when the blood is insufficient in amount duly to satisfy the capacity of the vascular system. The heart is thus like an empty pump, and is unable to drive on the blood. Here the obvious indication is to increase the volume of blood by the addition to it of some liquid, preferably normal salt solution, in order to enable the circulation to be carried on.

In place of intravenous injection two or three pints of normal saline solution may be injected into the subcutaneous tissue, preferably under the breasts. Rectal injection of warm saline solution is also an invaluable method, owing to the rapidity with which absorption of fluid takes place in the large intestine. In cases of severe hæmorrhage 1 to 2 pints of warm water injected into the rectum will be retained and rapidly absorbed. This method requires no assistants or sterilisation, and is only second in rapidity of action to intravenous injection.

Local Treatment of Hæmorrhage.—Primary Hæmorrhage.—Before describing the various measures which may be employed in order to control, either temporarily or permanently, bleeding from a wound, it is well to emphasise the fact that pressure properly applied to the bleeding point will suffice to stop temporarily bleeding even from the largest vessels whilst measures for the permanent arrest of the hæmorrhage are being undertaken. This is a point too often lost sight of.

1. *Position.*—Elevation of the arm or leg always diminishes and may of itself suffice to arrest hæmorrhage from the part in question. Forced flexion of a joint, elbow or hip, for example, on the proximal side of the bleeding point has a similar effect.

2. *Compression.*—This may be applied directly to the bleeding point, preferably by the fingers of the surgeon or an assistant, or the main artery may be compressed between the wound and the heart.

(*a*) *Digital Compression.*—As mentioned above, digital compression will temporarily arrest bleeding from any vessel, but can only be kept up for a comparatively short time.

(*b*) *Direct Pressure by Means of Antiseptic Compresses.*—This is a valuable method of treatment in cases where there is considerable oozing of blood from the wound, particularly in the case of a large cavity. The wound may be tightly stuffed with antiseptic gauze or wool, and over this a bandage, preferably elastic, should be somewhat firmly applied. The gauze packing should be left in position for two or three days, though if the bleeding be arrested the bandage may be removed earlier. This method is not to be recommended in the case of furious bleeding from a large artery.

(*c*) *Tourniquets* are instruments by means of which pressure is applied to the main artery on the proximal side of the wound. Their employment is an invaluable method of securing temporary control of arterial hæmorrhage, but cannot be continued for more than one to two hours owing to the extreme pain they cause.

Improvised Tourniquet.—Fold a handkerchief into a band 1 to 2½ inches thick; into the middle of this place a round stone or large round bullet, tie the two ends of the handkerchief loosely together after encircling the limb in such a way that the stone is placed over the main artery. Now insert a strong stick inside the encircling band of handkerchief and twist the stick until hæmorrhage ceases. This is a simple and effective emergency tourniquet.

Elastic Constriction (*Esmarch*).—An elastic band or cord forms the most useful tourniquet, as by its use the circulation in a limb can be entirely arrested. It is made to encircle the limb above the bleeding point. It should be applied with just sufficient firmness to control the hæmorrhage. If too tightly applied motor paralysis and laceration of muscles may be caused. An objection to this tourniquet is the fact that free capillary bleeding due to vaso-motor paralysis generally follows the removal of the constriction. The elastic band is greatly to be preferred to the cord-like or tubular constrictor.

Among other forms of tourniquet mention need be made only of two, Petit's and Lister's.

3. *Cold and Hot Water.*—Cold water just above the freezing point (32° F.) temporarily checks bleeding, but is considerably inferior to hot water. It seems unquestionably to increase the shock, and for this reason is in-

advisable in severe hæmorrhage. Hot water which has been sterilised by boiling, and then allowed to cool down to 130° to 120° F., is an excellent styptic for considerable oozing from large surfaces or cavities, though it is useless when the hæmorrhage comes from a large artery. By the employment of compresses soaked in water at 120° to 130° F. pressure and hæmostasis are at the same time obtained. Hot water seems probably, in great part by supplying heat, to diminish shock, and is therefore doubly indicated.

4. *Styptics*, as liquor ferri perchloridi, charpie, cobwebs, etc., were greatly used in olden days. Their employment is always inadvisable, and we mention them merely to condemn them as harmful and unscientific and dirty.

5. *Torsion* is a valuable and efficient method of arresting hæmorrhage in arteries which have been completely divided. The bleeding vessel is clamped with forceps, and a second forceps seizes the artery a short distance higher up at right angles to the vessel. The second pair of forceps holds securely the artery whilst a few twists of the first forceps in the direction of the long axis of the vessel suffice securely to obliterate the lumen of the artery.

6. *Acupressure.*—In this method a needle is passed under the vessel from the skin surface, which is twice pierced, and a figure-of-eight loop of silk is passed over the vessel around the two ends of the needle so as to exert compression. A more rapid method is to introduce the needle parallel with the artery, and then turning the needle through an angle of 90 degrees, pass it under the artery, which in this way suffers some degree of torsion as well as of compression.

7. *Suturing.*—Suture of a wound in an artery is not to be recommended owing to the tension in the vessel. It is a useful method, however, in wounds of veins. The suture should not penetrate the internal coat.

8. *Forcipressure.*—This is perhaps the most valuable method we possess, especially when used in conjunction with ligature of the larger vessels in the wound. Forcipressure is used in almost every surgical operation. Every bleeding point is clamped with artery forceps, which themselves suffice to arrest the bleeding in the case of tiny vessels. Any vessel, however, of any size requires to be ligatured or twisted. In certain instances in which, for various reasons, it is impossible to ligature a bleeding point, the vessel may be clamped and the forceps may be allowed to remain from two to four days, when the bleeding point will, except in very large arteries, be safely sealed.

9. *Cauterisation*, applied by the employment of Paquelin's cautery, the electric cautery, or any hot iron, is in certain cases a valuable means of arresting hæmorrhage, provided that the cautery be kept at a dull cherry-red heat. If used at a white heat the hæmostatic effect is lost. A disadvantage in cauterisation is the necessary production of a slough. The method, however, is valuable in not a few cases in which a ligature cannot be applied.

10. *Ligature.*—(*a*) With inclusion of surrounding tissues in certain cases in which, for any reason, *e.g.* the density of the tissues or the unhealthy condition of the vessel wall, the bleeding end of an artery cannot be clamped and ligated. A curved needle threaded with a ligature is then to be carried to some depth into the tissues around the bleeding vessel, which is sufficiently compressed by tying the ligature.

(*b*) *Ligature proper of the Vessel freed from surrounding Tissues.*—This is the method generally employed. Silk and chromic catgut are the materials which are generally to be recommended. Every substance employed as a ligature must be carefully sterilised. Silk can readily, by means of boiling for a few minutes, be rendered sterile, and this is its great advantage. Catgut, being made from the submucosa of the intestine of the sheep, is naturally most septic. It requires soaking for days in a strong antiseptic solution to ensure it being germ free. A great advantage of chromic catgut lies in the fact that it is absorbed in from one to three weeks. Thick catgut, however, is difficult to sterilise.

In applying a ligature the mouth of the artery should be clamped with forceps and drawn out somewhat from its sheath, and the artery tied with a reef knot.

It is safer in every instance to tie the ligature with sufficient force to ensure rupture of the middle and internal coats of the vessel. When an artery is completely divided, both the proximal and distal ends require ligation. Should an artery be incompletely divided, it should be cut right across after a ligature has been applied on either side of the bleeding point. The object in view in this procedure is to allow of retraction and contraction of the severed ends of the vessel.

TREATMENT OF REACTIONARY HÆMORRHAGE.—Unless due to the slipping of a ligature this amounts to nothing more than compression of oozing vascular points.

TREATMENT OF SECONDARY HÆMORRHAGE.—This is a subject of sufficient importance to demand special notice.

1. If the hæmorrhage is but slight it may suffice to reopen the wound, remove the blood coagulum, wash out with a hot antiseptic solution, and apply a compress or stuff the wound with double cyanide gauze.

2. Should the bleeding be severe, a tourniquet must be applied, or the main artery compressed digitally. The wound must be reopened and the bleeding vessel sought for and ligated. Should this be impossible owing to the softened, sloughy state of the vessel walls, the main artery must be ligated on the proximal side of the bleeding point by a fresh incision through healthy tissues.

3. Should this method fail, the artery may be ligated still higher up, or in the case of an extremity the limb may be amputated.

4. In cases of even slight secondary hæmorrhage it is a wise precaution to have, in the case of a limb, a tourniquet loosely applied above the wound, so that it may, in case of a sudden gush of blood, be readily tightened by the nurse in attendance. In places where a ligature on the proximal side of the bleeding point cannot be applied, the actual cautery may be applied and the wound packed firmly with antiseptic gauze.

Hæmorrhoids. *See* RECTUM, DISEASES OF.

Hair, Diseases of. *See* SKIN, APPENDAGES OF.

Hammer Toe. *See* DEFORMITIES.

Hand.

See also FINGERS.

SURGICAL ANATOMY.—The extensor tendons and superficial veins are seen on the dorsum. Situated respectively on the outer and inner sides of the palm are the thenar and hypothenar eminences, the former being composed of muscles acting on the thumb, the latter consisting of the small muscles of the little finger. The skin of the palm is thick, and contains neither hair nor sebaceous glands, but possesses an unusual number of sweat glands. The subcutaneous tissue in the palm is sparse, but the skin is well supported by the palmar fascia, and thus enabled easily to resist the pressure to which it is so frequently exposed. This fascia, too, affords protection to the underlying vessels and nerves. The superficial palmar arch crosses the palm in a manner represented by a curved line running from the pisiform bone, and the lowest part of the arch being in the middle of the palm at a level of the web between the thumb and index finger. The deep palmar arch lies a quarter of an inch higher up the palm.

APPEARANCES IN CERTAIN DISEASES.—In examining the hands one should notice whether there be any cyanosis or œdema, the latter being most noticeable on the dorsum. The strength of the grasp should be ascertained, and, if deficient, recorded by the dynamometer.

The *shape* of the hands may be abnormal in consequence of some general disease. Thus in *acromegaly* the hands, though not deformed, are much enlarged and extremely broad, the enlargement being mainly due to increase of the soft parts. The palmar creases are unusually distinct. In *pulmonary osteoarthropathy* the hands are enlarged, but also somewhat deformed, and the fingers are clubbed at their extremities. In *syringomyelia* the hands may be enlarged, but they too show deformity, for instance, muscular atrophy, contraction, and scarring. In *myxœdema* the dorsum is swollen, but does not pit on pressure and the hands are cold. The hands of a *cretin* are short, broad, and appear somewhat swollen, are sometimes of a purplish colour, and the skin is crumpled, redundant, and cold. In *achondroplasia* the hands somewhat resemble those of a cretin, but the ring and middle fingers are curved away from one another, and the skin is more natural than in the cretin. The metacarpal portion of the hand in *Mongolian imbecility* is small and soft, while the fingers, as compared with the hand, are somewhat thick, but taper at their extremities, and the little finger has often a curve, the concavity of which is towards the ring finger.

General atrophy of the hand is chiefly observed as a congenital defect or as a result of infantile paralysis. Wasting of the soft parts is seen in the course of general wasting diseases, starvation and old age, whilst atrophy, localised to certain areas, indicates some myelopathic or nervous lesion. Thus one sees atrophy of the thenar and hypothenar eminences in progressive muscular atrophy, and sometimes in syringomyelia and lead palsy. Localised atrophy may be associated with *contraction* and *deformity*, as occurs in ulnar paralysis from wasting of the interossei and the two inner lumbricales, the contraction affecting specially the little and ring fingers of one hand, or again in the *main en griffe* of progressive muscular atrophy, where all four fingers are equally involved.

The alteration in form may exist chiefly in the *joints*, which may be swollen, painful, distorted, etc. The causal condition will in most cases be rheumatism, rheumatoid arthritis, or gout, and in gouty subjects one may also find tophi, Heberden's nodes, and exostoses. In consequence of disease or injury of a peripheral nerve, the *nutrition of the skin* may be impaired in the area supplied by it, and the skin becomes thin and shiny, or is even the seat of other trophic lesions.

Fibrillary twitchings in the muscles are seen in several conditions, *e.g.* progressive muscular atrophy, amyotrophic lateral sclerosis or syringomyelia, but the first-mentioned of these conditions is the most frequent cause. As regards *tremors*, the most important, from a clinical standpoint, is the tremor so often seen in the hands in chronic alcoholism.

WOUNDS.—The hand is one of the regions of the body most frequently wounded, and the majority of the wounds are on the palmar aspect. A small punctured wound of the palm may be troublesome on account of hæmorrhage from a punctured vessel. This is best dealt with by enlarging the wound and applying a double ligature to the injured vessel. Fragments of glass, broken needles, and other *foreign bodies* often penetrate, and chiefly into the palmar surface. They may be followed by suppuration, or may only give rise to pain when the skin over them is touched. It is often difficult to locate with accuracy, and still more difficult to remove these foreign bodies.

SEPTIC INFLAMMATION may occur after a wound of the hand itself, or may spread to the hand from elsewhere. Thus it is common after a whitlow, and more especially after a whitlow of the thumb or little finger, in which digits the flexor sheaths continue upwards through the hand to above the anterior annular ligament. The symptoms are in general the same as those of suppuration elsewhere, but in the palm, owing to the density of the more superficial tissues, any deeply-seated inflammation is liable to be unusually painful, and if pus be formed, it tends to travel either above the wrist, towards the fingers, or towards the dorsum of the hand.

TENOSYNOVITIS, or inflammation of the tendon sheaths, may be seen affecting the flexor or extensor sheaths in the hand or the fingers. If it be acute, there is pain, stiffness, creaking on movement and sometimes fluctuation. Or the acute form may be of a suppurative character, and then tends to cause necrosis of the tendon and suppuration round the sheath. Pus in the palm travels to the regions previously indicated. Chronic tenosynovitis is usually tubercular, and occurs more often about the wrist than in the hand itself or the fingers. The affected sheath contains a variable amount of fluid in which one finds the characteristic melon seed bodies.

Compound palmar ganglion, which is usually a tubercular tenosynovitis of the common flexor sheath, is characterised by a chronic swelling in the palm and in the lower part of the forearm. This swelling appears to be divided by the anterior annular ligament into two portions, but the fluid can be driven from one portion to the other beneath the ligament. Simple ganglion, too, may be seen in the palm, but the condition is dealt with elsewhere. (See GANGLION.)

THE BONES of the hand may be affected by any of the ordinary bone diseases, such as periostitis, osteomyelitis, necrosis, or tuberculosis. Mention may, however, be made of *dactylitis*, which mainly occurs in the form of tuberculous disease of the phalanges in children, less often as a gummatous periostitis in syphilitic patients. The differential diagnosis of these two forms of dactylitis rarely presents much difficulty.

TUMOURS of the hand. The most important are the chondromata, cartilaginous tumours of benign nature. They are firm, rounded, often multiple, and may produce great deformity and uselessness of the hand.

Hanging, Death by. *See* MEDICAL JURISPRUDENCE.

Hare-Lip and Cleft Palate. *See* PALATE.

Hay Fever.

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THE STUDY of hay fever is a matter of recent years, the first good account of it being written by a Dr. Blackley of Manchester in 1873. He was a great sufferer himself, and wrote an admirable brochure on it. He was the first to substantiate the point that the commonest source of irritation is the pollen of certain grasses, especially of ripe rye-grass. He also noticed the fact that it was only the few who experienced any discomfort from it, though all were equally exposed to it. The next writer on the subject was Dr. Beard of New York, who, five years after, pointed out that nearly all the patients were of the neurotic temperament, and that constitutional treatment directed towards the amelioration of this condition was often of considerable value. Then Dr. Marsh, also of New York, in 1877 published a paper on hay fever. He himself was susceptible only to the pollen of the common rag-weed (artemisia), which blossoms only in the autumn in America. But he was obviously mistaken in supposing such patients were susceptible to this alone, as in that case the hay fever of those suffering in the spring would be unaccounted for. In the year 1882 Dr. Daly of Pittsburg contributed perhaps the most important theories of all, though he in his turn was practically misled. He maintained that hay fever was invariably due to morbid conditions of the nose, and that they could all be successfully treated by intra-nasal operations. But cases are frequently encountered in which the nose is absolutely normal, and such would be inexplicable on this supposition. Sajous in the following year made a contribution of some value, in which he corroborated Daly's theories, which were subsequently embraced by specialists all over the world—in Vienna by Hack, and in this country by Woakes, M'Bride, and many others. But Dr. J. N. Mackenzie of Baltimore advanced the theory that the coryza, as he considers it, is dependent upon some functional derangement of the nerve-centres rather than any abnormality in the mucous membrane. Then there appeared Morell Mackenzie's able treatise in 1885, in which he contended that there was no intra-nasal disease at all, and that the whole trouble was an idiosyncrasy dependent upon the general health. All these writers were practically right; and yet not one of them, as appears to me, has grasped the subject from a wide point of view. I think we may define hay fever, in the first place, as an idiosyncrasy which renders certain individuals abnormally sensitive to sources of irritation to which they, in common with the rest of their fellow-creatures, are exposed; which idiosyncrasy may be augmented, if not actually started, by abnormal conditions of the nose. Yet it must be admitted that we find many a case in which the mucous membrane is exquisitely sensitive to these unwarrantable sources of irritation, and, nevertheless, in which we find no morbid condition whatever in the nose. Yet, again, although there may be abnormalities in the nose, they do not justify our saying that the hay fever is actually caused by such abnormal conditions. Rather may we assert that when the affection has persisted for many years it may actually lead to pathological conditions.

Now I must enlarge a little further on these points. We may have abnormalities in the nose interfering with respiration, yet in themselves not necessarily pathological, but merely malformations. We may have many forms of obstruction to respiration from hypertrophy, such as spurs and deviations of the septum. These are common causes of obstruction, and when they occur to any extent they more frequently obstruct the inferior meatus. And it seems to me, rather from clinical observation than from any theoretical consideration, that it is essential that these patients should breathe easily through the inferior meatus. In all patients there is a strong instinct to breathe through the nose rather than through the mouth, often in spite of grave obstruction, though perhaps the instinct does not assert itself so forcibly in adults as in children. When the inferior meatus is obstructed, the inspired current of air, instead of passing along the less sensitive portions of the nose, is drawn up into the middle meatus, which is more sensitive and irritable than the lower. So that in many cases of hay fever the history is this: at the starting-point we have the idiosyncratic hyperæsthesia, the consequence of which becomes more serious if the inferior meatus be obstructed; next, as the consequence of the continued irritation, actual hyperplasia of the inferior turbinated bodies is induced. Yet we must admit on clinical grounds that these cases of real hyperplasiæ offer as good a prognosis for surgical treatment as those in which the obstruction is structural rather than pathological.

As to the conditions under which the affection may begin, we have predisposing causes as well as those immediately exciting. In the first place there is *climate;* hay fever occurs in temperate latitudes, being in tropical countries almost unknown. Then we find that the idiosyncrasy is confined to certain *races*, being especially found among the Anglo-Saxons and the associated portions of the Celtic race: the Irish are certainly not exempt. *Heredity* is a factor of the greatest importance. I have in my books the case of a mother and two daughters who suffer from hay fever; a third daughter suffers from paroxysmal sneezing all the year round, although she does not have hay fever; one son suffers severely from hay fever, and another from nasal asthma—that is to say, bronchial spasmodic asthma cured by intra-nasal treatment. All these cases presented abnormal conditions of the nose. Sajous maintains that in 35 per cent of all his cases there was well-marked evidence of heredity, and that 42 per cent had asthmatic relatives. Next we find also that *class* has much to do with the tendency to sneeze—the educated classes being much more prone to the affection than the uneducated. Mackenzie said he never had a case among his hospital cases; but the hospital class is becoming better educated and more neurotic every day, and we certainly now find among them a fair proportion of hay fever cases. Then *town-dwellers* are more prone to it than country gentlemen, though I have seen several instances of it among the latter with apparently normal nerves and plenty of good blood. *Sex*, too, makes a difference, men, it is said, being more prone to the affection than women. Among other causes of predisposition, the long existence of catarrhal conditions renders patients more sensitive to those forms of irritation which occur in the summer. Of the direct sources of irritative attack of course the pollen of grasses, especially of rye-grass, is the most common, beginning to harass its victims about the first week in June, often much earlier, according to the degree of advance of the season. Some begin sneezing so early in spring that one is compelled to accuse the catkins; while the ragweed, which is confined to America, does not begin its attack until the autumn. Other patients are sensitive to nothing but roses. While the hay-fields are the misery of most patients, less commonly the moors and heather will make some men suffer when they go grouse-shooting. Others begin sneezing when exposed to the bright sunlight at sea, though the latter gives immunity to many who suffer from vegetable sources of irritation. There is a plant in

Australia which induces sneezing; it flourishes in the spring, about September, and I believe it is commonly known as the cape-weed. It covers the hills around Adelaide to the height of some thousand feet or so. It is a composite, and the pollen is so profuse that after driving for two or three miles in the country the sides of the carriage will be covered with its yellow dust. I am told that most of the population of Adelaide are affected with hay fever during the time of the blossoming! Certain other people, again, only suffer from animal sources of irritation. One patient tells me she always sneezes when a cat comes into the room; and I know several who suffer from sneezing when in contact with horses.

The symptoms usually begin with a violent paroxysm of sneezing on waking early in the morning, sometimes supervening suddenly and sometimes more gradually. Occasionally, for many days before it actually begins, the patient will experience a feeling of irritation about the inner canthus of the eye, which he is compelled to rub continuously, or an itching about the alæ of the nose. In one of my patients the earliest symptom is a coldness and pallor of the nose, which, though it is warm sunny weather, he has to rub to restore the circulation. Sometimes the sneezing will last only for a few minutes, sometimes for many hours, and it is always accompanied by a profuse flow of watery mucus from the nose. A patient will sometimes assert that he saturates a couple of towels in the space of half an hour, after which he is naturally quite exhausted; indeed the collapse that sometimes follows is very distressing. Seldom are patients seen in greater misery than those suffering from hay fever—strong men becoming anæmic wrecks after six or eight weeks of the malady, which time is the ordinary duration, although many patients remain prostrate for as long a period after the symptoms have subsided. Of course if they go out of doors during these weeks the symptoms become aggravated, especially if there is wind and sun; so that they have to protect themselves in a very extraordinary way—with thick veils of gossamer over their faces, blue spectacles, and green umbrellas! Some of these patients suffer from more or less sneezing all the year round, these being especially cases where the continual attacks of hay fever have induced actual hypertrophy of the inferior turbinated bodies. Sometimes a patient is found who is sensitive to every source of irritation, not only to pollen in its various forms, but to emanations from animals. They suffer continually from dust; they cannot take a book from the bookshelves for fear of provoking sneezing; they dare not go near a feather bed or into a room after it has been dusted; and so on. The slightest changes of temperature will cause some swelling of their Schneiderian, adjacent surfaces will come into contact, and thus may the same train of symptoms arise even without the introduction of any irritating substances.

Usually the asthma of hay-fever patients is a later symptom; the sneezing has persisted for ten days to three weeks before it begins, and sometimes the nose-symptoms abate when the asthma is fairly established. This probably occurs as soon as the swelling in the nose becomes so pronounced that nasal respiration is no longer possible, the bronchi themselves becoming thereby more exposed to the pollen, etc. Yet I must confess that many patients suffer from asthma alone without any sneezing symptoms at all; and it seems to me that these are more often cases of permanent obstruction to the nasal respiration.

A few words on prognosis must preface those in reference to treatment. In cases where we can find neither structural nor pathological abnormalities, the prognosis is bad. All we can hope to do for such patients is to mitigate their symptoms as much as possible. The cases that are best are those in which we find something demanding operation, and the bigger the obstruction the better the prognosis. The actual duration of the disease, whether the patient has suffered for few or many years, does not affect prognosis in any way. The most striking case of cure I have had was, without exception, the worst case in my experience. The patient, a man of intensely active mind and neurotic temperament, had for thirty years considered the whole of the summer a misery; yet he was completely cured by the removal of a large spur on the septum completely blocking the inferior meatus; and for ten years, so far as I know, he has had no return of symptoms. I do not think, either, that the general condition of the patient affects the prognosis; although it is of some consequence whether or no he be very neurotic. To differentiate a little further, the most suitable cases for surgical treatment are those in which we find the inferior meatus obstructed, whether by deviations or spurs of the septum, or by true hyperplasia of the erectile tissue; next to these come chronic engorgement of the inferior turbinated; next adenoids in children. Less amenable are cases of polypus obstructing the middle meatus; and it is the fact of having seen polypi in typical hay-fever cases, where the removal of the growths gave but little relief, which has inclined me to the belief that the restoration of breathing through an obstructed inferior meatus is the essential factor in treatment.

Further, we find cases in which there is no actual obstruction in either inferior or middle meatus. Opposite to the anterior extremity of the middle turbinal we often find on the septum curiously ill-defined œdematous and pallid swellings which obscure the view, and bring the septum into contact with the middle turbinal. Like engorgement of the inferior spongy bodies, they are boggy, and pit under pressure, while they collapse under cocaine in a few seconds. Destruction of these boggy swellings with the galvano-cautery is often attended with the happiest results. Presumably in these cases the trouble is due to direct irritation, which induces swelling of the mucous membrane where it abuts on the middle turbinal, thus evoking the whole train of symptoms. Such, on the whole, are good cases to treat. Let me repeat once more that the worst cases for treatment are those where we can find no structural or pathological abnormality to deal with. Where there is opportunity for operation we should advise it, although it is impossible to be absolutely sure of effecting a cure; indeed we seldom attain an *absolute* cure. Yet in this disease, as in all others of the nose, let us, in determining whether or no operation be necessary, remember M'Bride's dictum that we should judge the nasal fossæ by what they can do rather than by structural aberration from the strictly normal.

In cases where surgical treatment does not promise much, what can we do? The first and most common remedy is only mentioned as a warning against its use: it is cocaine. The mischief it induces is sometimes appalling, and the patients at the end of the summer are generally worse than if they had not used it at all. But the temporary relief it affords is unquestionable. Patients may begin with a 2 per cent solution, but are soon compelled to increase the strength rapidly, until they often end with a 30 per cent solution; and, finally, they become wrecks from the pernicious effect of the drug on the nervous system. I have seen two such cases in medical men, one of whom appeared to be developing symptoms of general paralysis, although he completely recovered as soon as he relinquished the habit.

But there are other local remedies of distinct help: the old-fashioned compound tincture of benzoin, the bismuth and morphia insufflations, known as Ferrier's snuff, both may give relief, and, on the whole, Ferrier's snuff is preferable to cocaine, although it does not arrest symptoms as decidedly. Sprays and lotions of borax, or boric acid, especially if combined with liq. hamamelis, are very soothing, and wash away accumulated secretion. The patient always likes such applications, though they must be used in very weak solution. A good method of application is for the patient to take the solution in a teaspoon, to hold the head back, and simply pour the warm fluid into the nose.

Another remedy I have occasionally found singularly useful has been a solution of chromic acid, $\frac{1}{16}$ or $\frac{1}{8}$ of a grain to the ounce, sprayed or poured into the nose three or four times a day or oftener, and held in the nose as long as possible. I had one patient who would carry about her spray of chromic acid just as others will carry about their cocaine. Another patient, after two or three weeks' use of it, seemed to be completely cured, and had no return of the symptoms the following summer. A good deal was talked a few years ago of using bichloride of mercury. Dr. Carl Genth used it in a $\frac{1}{3000}$ solution dropped into the eye, and allowing it to pass into the nose. I have heard two or three practitioners speak highly of it, and I have every reason to think it worth trying. Strong carbolic acid has been much vaunted as a cure; but I have seen so much mischief accrue from its incautious use that I cannot recommend it—at any rate so long as we have safer and more certain means of treatment. A 5 per cent solution of eucaine hydrochloride (A) sometimes gives great relief, though its application stings at first, and it does not act so strikingly as cocaine. But it does not exhibit the physiological disadvantages of the latter drug. Menthol sometimes relieves; and the following prescription I have found of the greatest service:—℞ Menthol gr. 5, eucaine (A) hydrochlor. gr. 5, zinci oxidi gr. 20, adipis lanæ ℥ss., paraff. liq. ad ℥j. M. Ft. ung. Sig.: To be applied to the nostrils frequently with a camel-hair brush. During the last few months I have found supra-renal extract 4 to 10 per cent as a spray of signal use in a few obstinate cases of ordinary paroxysmal sneezing; and I suspect it will prove beneficial to some sufferers from hay fever.

Another point of importance in treatment is the careful feeding and resting of the patients, while they are probably all better for some stimulant. No special dietary is indicated seeing that the patients present no tendency to lithæmia, etc. Various nerve tonics are also useful, like nux vomica, valerian, and asafœtida. Rather than give the patient cocaine, it might be wise to allow the opium pipe. Its use is risky, as patients grow addicted to it, and I have never had to prescribe it; but its power of controlling the worst symptoms is beyond all question.

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Head.

See also BRAIN,[1] SKULL, SCALP.

THE *shape* of the head may be quite normal on examination, but there is often a definite abnormality produced by certain diseases.

In childhood a departure from the normal shape may be due to several causes. In well-marked *rickets* defective ossification is a prominent feature, the sagittal and coronal sutures remain open after the fourteenth month, the fontanelle does not close during the second year of life, and in consequence of defective ossification and thinning of the bones there may be areas which give on pressure and yield to the fingers a sensation of "parchment crackling." This condition is called cranio-tabes, and is usually most marked in the parieto-occipital region. If rickets lasts for some time there will be hyperostoses on the outer surface of the cranial bones, especially the frontals and the parietals, causing what are known as nodes or bosses, and the head becomes characteristically square. Neither the bosses nor cranio-tabes, however, are pathognomonic of rickets, as they are found in *congenital syphilis*, and are especially pronounced in those children who are the subjects of both these diseases. In congenital syphilis there is depression of the bridge of the nose, fissures about the mouth, Hutchinson's teeth, and other signs which do not occur in rickets. In *chronic hydrocephalus* the head is large and globular, the eyeballs are prominent from depression of the orbital plates, the superficial veins of the scalp are distended, the face is proportionately small, the head is with difficulty held upright, the sutures and fontanelles are widely open, and there is cranio-tabes. In *microcephaly* the head is small, the vertex tends to be conical, the forehead recedes very markedly, and the sutures and fontanelle close early. In *cretinism* the head is large as compared with the body, the forehead, as in microcephaly, is low and receding, but the anterior fontanelle remains open for years, and the eyes are wide set from one another. In *Mongolian imbecility* the head is short and spherical, and the eyes are set somewhat close to one another, while in *achondroplasia* the head is large and broad, the forehead is prominent, and the nose resembles that of a bulldog.

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[1] The subjects of concussion and compression are considered in the article on the Surgery of the Brain in vol. ii.

In *acromegaly* the cranium is often large, and the sagittal suture is palpable as a thickened ridge. The facial alterations are more pronounced, and consist chiefly in enlargement of the zygoma and frontal processes, and especially of the inferior maxillæ, whereby the face is much lengthened. Enlargement of the skull is also seen in *osteitis deformans*, but here the facial bones are hardly ever affected; and also in those rare conditions known as *diffuse hyperostosis*, which affects the skull bones and occurs in youth; and, lastly, in *leontiasis ossea*, in which disease there are hyperostoses in the form of tumours growing from the skull bones, and especially the maxillæ, the disease commencing in childhood and producing great deformity in later life.

SYMMETRY.—The cranium of infants is often somewhat asymmetrical, either without definite cause or in consequence of rickets. Asymmetry of the head may be seen in *hemihypertrophy*, or specially affecting the face, as in *progressive facial hemiatrophy*, or more frequently in association with wry-neck, where there is imperfect development of the face on the same side as the affected sterno-mastoid.

In babies the anterior fontanelle normally closes between the fifteenth and eighteenth months. The conditions which retard closure have been already mentioned, *e.g.* rickets and chronic hydrocephalus. Examination of the fontanelle shows that its normal tension may be raised by crying or coughing, or by cranial tumours, meningitis, or hydrocephalus, which produce increased intracranial pressure, while the most common cause of depression of the fontanelle and lowering of its tension is severe and continued diarrhœa. The fontanelle has normally a slight pulsation communicated to it from the arteries forming the circle of Willis, but auscultation is useless in determining the presence of intracranial disease.

The percussion note of the skull is of doubtful value in diagnosis, but percussion is often helpful in locating such conditions as cerebral tumours and meningeal gummata, or in forming a diagnosis of mastoid inflammation.

Head retraction, or cervical opisthotonos, is an important sign in babies of meningitis, whether of tubercular or of the so-called simple form. In its mildest form there is merely rigidity of the neck, due to tonic spasm of the muscles at the back of the neck, but when well marked the head is fully extended, the occiput even touching the back, and in such a case the inflammation has probably spread to the spinal meninges. Head retraction is also seen in infants with chronic diarrhœa, or associated with cerebro-spinal congestion, *e.g.* in typhoid fever. The condition must not be mistaken for wry-neck, or the stiff neck of rheumatism or spinal caries. In adults we find head retraction in tetanus and hystero-epilepsy.

Conjugate deviation of the head and eyes is due to cerebral hæmorrhage, meningitis, or tumours, but most often to hæmorrhage. The eyes and head are turned towards the side where the lesion exists, *i.e.* away from the paralysed side; but in the stage of early rigidity they may be turned in the opposite direction. In hæmorrhage of the pons the deviation is just contrary to that of other hæmorrhages.

Injuries of the Scalp; Scalp Wounds.—The edges of the wound do not gape if the cranial aponeurosis be uninjured, and the appearance of a wound inflicted with a blunt instrument will closely resemble that of an incised wound. If the aponeurosis has been divided there is much gaping, and the scalp can be freely stripped off from the pericranium. Scalp wounds usually give rise to severe hæmorrhage, because the vessels which lie in the fibrous tissue of the scalp are unable to retract. The wounds heal readily. The chief dangers are hæmorrhage and suppuration in the space between the pericranium and the aponeurosis of the occipito-frontalis. If the pericranium has been detached from the bone by violence or suppuration there is merely slight superficial necrosis of the bone, which is hardly in any way dependent on the pericranium for its blood-supply. It is of extreme importance to ascertain whether there is a fracture of the skull in addition to the scalp wound. Thorough investigation of the wound is imperative, and if in doubt it is a good rule to enlarge the wound of the soft parts, and explore with the aseptic finger, so as to avoid mistaking torn pericranium for a fracture, as one may do if a probe be used. Of less importance as local signs of fracture are (1) an infiltration of blood into the conjunctiva and eyelids, most commonly seen in fractures involving the orbital plate or the orbital ridge, and which must not be mistaken for the infiltration following a local injury to the eyelids; and (2) the escape of blood or cerebro-spinal fluid from the ears, mouth, or nose after fracture of the base.

Cephalhæmatoma is a blood tumour of the scalp caused by trauma. According to its position in the scalp we speak of it as (1) subcutaneous, (2) subaponeurotic, or (3) subperiosteal. The hæmatoma may form a well-defined swelling, which, with a soft and somewhat depressed centre and a firm, elevated margin, may resemble a depressed fracture. The diagnosis of hæmatoma is determined by exerting pressure on the elevated margin, which is then found to disappear.

Subaponeurotic hæmatoma is not common, but if it occur the blood is only limited by the attachments of the occipito-frontalis. The subperiosteal variety is confined by the pericranium to one bone, and is most often seen in infants over the right parietal bone after forceps delivery.

The treatment of scalp wounds requires no special mention, further than emphasising the importance of thorough cleansing and shaving of the scalp for a considerable area around the wound.

Cephalhæmatomata are best left alone, for the blood will be absorbed.

Strapping, pressure, evacuation of the blood, or incision and drainage on account of suppuration, are rarely required.

Tumours of the head are mostly seen in the scalp. They may be simple sebaceous cysts, or dermoid cysts. The latter are most common about the outer angle of the orbit, yet may be found in the middle line, and then closely resemble meningocele. Epithelioma, fibroma, the various forms of angioma, and more particularly the so-called cirsoid aneurysm, and sarcoma may be mentioned. By cephalhæmatocele is meant a cephalhæmatoma, which communicates with one of the sinuses of the dura mater, causing a small reducible swelling, which may pulsate. Lastly, there are the congenital malformations of the skull, causing cephalocele, the most common variety being the meningocele, which, as a rule, occurs in the middle line, and is more or less reducible and translucent.

Diseases of the Bones.—Osteomyelitis occurs as a result of compound fracture (*q.v.*). Tuberculosis of the cranial bones is a very chronic condition, in the course of which a tubercular abscess forms. This bursts, and sinuses remain running through the bone.

Syphilis gives rise to gummata in the pericranium or bone, or between the bone and dura mater. They either disappear under treatment, burst externally, or become ossified, and then form "syphilitic exostoses." The head in congenital syphilis and in other general diseases has been already referred to.

Headache.

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HEADACHE has been aptly described by Handfield Jones as a *Dis-ease.* It is a symptom of a great variety of morbid conditions, and frequently the discovery of its cause is a problem of great difficulty. In all cases the pain is perceived in the sensorium. The seat of its production may be: (*a*) nerve-endings, in the meninges, the scalp, some organ of special sense, or, at a greater distance, in the stomach, or some other organ; (*b*) irritation at any part in the course of a nerve; (*c*) or it may arise from the condition of the nerve-cells of the sensorium itself, or of the blood surrounding them.

The CAUSES of headache have been divided into *organic* and *functional.* The investigations of Hodge on the changes produced by fatigue in the nerve-cells of the bee render it probable that, with improved methods and more exact knowledge, the class of functional disorders, with the exception, perhaps, of those due to reflex irritation, will disappear from our nomenclature. As a factor in the production of headache, the quality of the sentient mechanism of the individual plays a most important part; some persons inform us that they never had a headache in their life; in certain individuals a small elevation of temperature is always accompanied by pain in the head, whereas high temperature causes no headache to others. Women are more liable to this kind of suffering than men; it is more frequent in persons of nervous temperament than in others. Age brings often a general blunting of the sensibility both of nerve-centres and of end-organs, and headache, as a rule, recurs with diminishing frequency and severity as life advances. Occasionally more than one cause operates in the same case; *e.g.* in fevers both hyperhæmia and toxæmia act.

The causes of headache may be arranged in the following order:—

1. *Intracranial Disease.*—Acute and chronic inflammations of the meninges from syphilis, gout, rheumatism, tubercle, chronic alcoholism, chronic kidney disease, general paralysis, and other forms of insanity. Head injuries occasionally produce headache for a long time after the

injury, although there may not have been external mark. Encephalitis, hydrocephalus, intracranial hæmorrhage, abscess, tumour. Small hæmorrhages and slowly-growing tumours in the substance of the brain are not accompanied by pain. Bramwell points out that tumours cause pain by (*a*) increase of intracranial pressure; (*b*) direct involvement of the membranes; (*c*) direct implication of the fifth nerve.

2. *Cranial and Pericranial Affections.*—Any disease which produces pressure on nerve-trunks or twigs as they pass through foramina in bone or membrane—syphilitic, rheumatic, and malignant diseases of bone or of pericranium,—erysipelas, wens, excessive weight of hair, certain styles of coiffure, pediculi, tight or too heavy head-dress.

3. *Reflex Irritation.*—In addition to some causes named in class 1, and most in class 2, there are those from the organs of special sense, and the abdominal organs. Irritation of the end-organs of special sense is a powerful exciting cause of headache because of the extreme sensitiveness of these structures.

(*a*) *Nose.*—Any disease of the nose and of its accessory cavities. Dundas Grant says that adenoids in the naso-pharynx are the most common form of nasal disease producing headache. Acute congestion of frontal sinuses in colds and in influenza cause severe frontal headache. Treatment of chronic disease of frontal, antral, ethmoid, and sphenoid sinuses has been followed by cure of chronic pain in head.

(*b*) *Eye.*—Muscular and retinal strain in a healthy eye, as by a visit to a picture-gallery; also the effect on the retina of glare from the sea, sand, snow, bright roads. Chronic or frequently-recurring headache may be induced by organic diseases of the eye, such as glaucoma and iritis. In chronic headache the ocular tension should always be carefully tested. Errors in the optic apparatus which induce fatigue are very frequent causes of headache:—1. These may affect the focussing: (*a*) errors of refraction—hypermetropia and astigmatism, less frequently myopia and anismetropia; (*b*) weakness of the ciliary muscle—following diphtheria, due to presbyopia, or to debility. 2. Errors in powers of fixation—esophoria, exophoria, hyperphoria—due to an excess or to a deficiency in the action of the muscles acting on the globe.

(*c*) *Teeth.*—The eruption of teeth both in first and second dentition; also of the wisdom teeth. This is a more probable cause when the jaw is small and the teeth impacted. Caries specially of teeth in the upper jaw.

(*d*) *Ear.*—Wax in the meatus, otitis media, cholesteatoma in the mastoid antrum. Legal points out that sometimes ear disease causes not so much pain in the ear as in some other part of the head.

(*e*) *Digestive tract.*—Occasionally we find that headache comes on after, or is aggravated by, the taking of food, and persists till the stomach is empty. Dilatation of stomach causes headache, probably through the production of ptomaines. Overloaded bowels may act in the same way, but overloaded bowels and rectum and hæmorrhoids also act by reflex irritation. Lumbricoids and tape-worm should also be borne in memory as causes in obscure cases.

(*f*) *Reproductive system.*—Although Matthews Duncan stated that the generative organs do not cause pain above the breasts, it is established that congestions and displacements of the uterus do occasionally give rise to headache. That which is frequent in young women about puberty and in older women at the climacteric is more directly related to the condition of the nervous system.

4. *Toxæmia.*—(*a*) Autogenetic, from uræmia, liver diseases, gout,

rheumatism, oxaluria, constipation; (*b*) Heterogenetic, from alcohol, impure air of crowded meetings, badly-ventilated bedroom, bad drainage, certain drugs, such as opium, quinine, etc., malaria, fevers. It is to be borne in memory that the liver, the largest gland in the body, stands at the gateway between digestion and circulation, and in deranged conditions is liable to permit the passage of ptomaines. It is often deranged without prominent digestive symptoms.

5. *Hyperæmia.*—Acute congestion in encephalitis, plethora, menstrual suppression, increased action of heart, violent exertion, and mental excitement. Passive congestion from certain diseases of the circulation and pressure of tumours on great veins leading from the head.

6. *Anæmia and Nervous Debility.*—Chlorosis and the various diseases which produce general anæmia induce debility of nerve-centres, and in some constitutions headache results. Overwork, prolonged lactation, exhaustion, mental anxiety—which Laycock called phrenalgia: he considered depression of spirits and hypochondriasis to be varieties of phrenalgia—neurasthenia, hysteria, sunstroke.

7. *Migraine* or *megrim.*—This is a paroxysmal form of headache allied to epilepsy and other nerve-storm diseases. As it differs materially from other forms of headache, it requires notice at greater length. It is hereditary, occurs in families of nervous constitution, in those who are intellectual and studious, and in members of families with a history of epilepsy, chorea, insanity, and nerve weakness. Although there is a case on record which commenced at the early age of three and a half years, it most often begins at puberty, diminishes in frequency and severity with advancing age; it generally ceases in old age, but not invariably so. About the menopause it generally becomes temporarily aggravated. The attacks are periodical—once a week or at longer intervals; in women it frequently occurs at the menstrual period, commonly just before it. An attack may last a few hours or several days, most frequently one day. Its onset is occasionally indicated by prodromata—in some persons a feeling of well-being, in others malaise, or sleepiness or constipation; frequently there are optical phenomena—dimness or confusion of vision, coloured lights, flashing, dazzling, hemiopsia, or teichopsia. The attack generally comes on in the early morning, either immediately on awaking or soon after, with pain or throbbing in one or both temples; there may be pallor or flushing of the face, or both may occur at the same time. The pulse is slow in proportion to the severity of the pain, the body is cold, the head hot. In severe cases the subject must rest—generally lying, but some must sit—in a darkened room; the pain is continuous with occasional exacerbations, which may be agonising. After a time nausea and vomiting occur, and following this the subsidence of the attack; in others vomiting recurs and is bilious; rarely diarrhœa is a symptom. During the attack the urine and saliva are diminished, and usually there is anorexia. Haig states that during the prodromal period of well-being the excretion of uric acid is below the normal, and that during the seizure it rises above it. Many theories have been advanced as to the cause of migraine. Haig believes that uric-acidæmia is to blame; some consider that the primary error lies in the digestive system, and in order to cure an attack and prevent recurrence treat the stomach; others locate the error in the circulation; others in the nervous mechanism. Uric-acidæmia does not produce migraine in every subject, only in those with a peculiar nervous system. The circulatory phenomena, the headache, sickness, uric-acidæmia, are not the disease, but incidents in the attack. In migraine, as in other nerve-storm diseases such as epilepsy, there are

probably at least three factors. 1. There must be a peculiar constitutional condition of the nerve-centres. 2. A morbid or toxic condition of the blood. 3. Some peripheral irritation. This irritation acts as the exciting cause; it may be worry, excitement, overwork, powerful impressions on any of the special senses, errors in the optical apparatus causing eye strain, digestive derangements.

8. *Electrical* and other conditions of the atmosphere produce headache in certain persons.

DIAGNOSIS OF THE CAUSE.—This is often a matter of great difficulty, sometimes all that can be attained is a provisional diagnosis. In the solution of the problem we first consider the age of the patient and the diseases common at the time of life, the circumstances preceding the attack, the time of day at which it occurs, the character of the pain—continuous or paroxysmal—and its duration. The locality of the pain may give some guidance, but it often gives none. We then make a systematic examination of the special senses and of other organs which might produce reflex irritation, also of the blood. The temperature and other symptoms enable us, as a rule, at once to put aside specific fevers and encephalitis, although occasionally in typhoid fever headache and debility are the symptoms which cause the sufferer to seek advice.

Age.—When we have to do with young children we should examine the gums. If the mouth be moist and the child do not suffer increase of pain when the gums are pressed on, dentition is not at fault. We should inquire as to accident, for headache may recur long after injury and in the absence of external mark. Examine the sutures for evidence of hydrocephalus. If the head be drawn back think of basal meningitis. In tubercular meningitis pain is not an early symptom; when it occurs it is in paroxysms, the child screams, may put its hands to the head, there is vomiting, constipation, marked leucocytosis, and the eyes if they can be examined show evidence of optic neuritis. Always when you suspect pain in the head of a child examine the ears for evidences of inflammation of them, by making gentle traction on the auricles, pressing on the tragus; and if possible examine the meatus and membrana tympani. In older children examine the ears, ascertain whether there has been discharge from the meatus; pain from inflammation of the meatus or of the middle ear is relieved after discharge, if not relieved is there abscess? Absence of tenderness on tapping the cranium, and behaviour of the temperature will guide. During second dentition caries and impaction of teeth are possible causes, the latter specially if the jaw be small. Lauder Brunton states that in every case of headache he first examines the teeth. If the child be of school age the condition of the optic apparatus should be examined as to errors of refraction and of powers of fixation, the nose and particularly the naso-pharynx are likely causes at this age. The attention should be specially directed to the latter if there has been frequent ear trouble, examine the naso-pharynx with the finger. If these and other physical causes such as irritable stomach and bowels can be excluded, ascertain the amount of school work which is required. If sleep be disturbed and the child awake unrefreshed, it has probably too much work. Overpressure is a relative term.

The period of eruption of the wisdom teeth is a very variable one, and the irritation connected with the process is a common cause of headache. Megrim often commences about puberty, and the history of the attack is diagnostic, unless in very mild cases. In later life it might be confounded with gouty headache, but the latter is usually confined to one side of the head, is much aggravated by movement; megrim usually becomes less severe in late life, the head may or may not be tender on tapping, there is no history of periodicity, but there is a history of gout in the individual or family, and there may be other evidences of gout present. About middle life a very common cause of headache is commencing presbyopia; if there be increased ocular tension we should be suspicious of glaucoma. In later life a not uncommon cause is uræmia. In every case of headache the urine should be examined.

The Time of Day.—If headache come on in the morning examine for uric-acidæmia, ascertain whether the bedroom be properly ventilated, and whether the drainage be good. Headache which occurs on awaking in the morning, and is relieved after breakfast, is often due to want of food, nothing having been taken since late afternoon, and then only a light meal. Some congestive headaches are aggravated by the recumbent posture with low head, and are therefore worst in the morning. When the attack comes on in the evening it is often caused by overwork, exhaustion, nerve debility, sometimes by commencing presbyopia. Those which come on or are worst at night are due to rheumatism or syphilis. When the latter is the cause the pain is more constant and the nocturnal exacerbations are, as a rule, more severe than in rheumatism. The headache of rheumatism may be very severe, but it is not so constant as that due to syphilis or tumour; there is usually the history of a draught, sometimes great tenderness of the scalp, and there is the history of former rheumatic ailments. Occasionally when the structures at the back of the head are affected in rheumatism the slightest movement of the head is painful. The diagnosis of intracranial tumour in early stages presents considerable difficulty, specially if it be slowly growing and if it do not involve any trunk of a peripheral nerve. The difficulty is all the greater from the fact that occasionally hysterical symptoms are the most prominent features in the early stages of organic disease. The characteristics of headache produced by tumour are, the constancy of pain with severe exacerbations, which may come on at night but are not confined to that period, and the pain seems to be present even during sleep. Gowers lays it down as a rule that headache which prevents sleep is due to organic disease, but the rule is not an invariable one. If in such a case examination of the fundus of the eye reveal symptoms of intracranial pressure, tumour may be diagnosed. Hæmorrhage as a cause of headache is excluded by the history. Abscess is often difficult of diagnosis, history of disease of ear, bone, or of pyæmia, with variable pulse and temperature, should guide. In encephalitis severe headache is the first symptom, it increases from hour to hour, and the temperature is elevated. It is not always easy to diagnose hysterical headache; usually the description given is out of proportion to the evident condition, the patient sometimes smilingly descants on the suffering; a common seat of this headache is the vertex; the pain is likened to the driving in of a nail. Violent exercise, laughter, coughing, blowing wind instruments, stooping aggravate congestive headache. If the condition be produced by fatigue, noise, bustle, study, it is probably due to debility or exhaustion. The amount of debility may be gauged by the facility with which the pain is induced,—the more readily induced the more tedious the recovery. If a nursing mother complain of headache after having given the breast to her child she should at once wean it. If headache be produced by sightseeing we have to do with a person debilitated, or naturally delicate, or with eye trouble.

Occasionally the condition of the pulse will put us on the track of the cause. If it be one of high tension, examine for toxæmia, uræmia, uric acid, or some other form autogenetic or heterogenetic. Liver derangement is a not uncommon cause of such trouble.

The *locality* to which pain is referred is not of much aid in diagnosis. Headache from eye strain, although often frontal, may be situated in any part of the head. Pain from ear trouble may be in any part of the same side, even on the opposite side; the ear itself may be free. Removal of cerumen has cured pain over the whole head. Ocular headache is common in biliousness, also in gout. Frontal headache is common in malaria, in dyspepsia, in congestion of the frontal sinuses, in catarrh and influenza, and in inflammations and suppurations of these sinuses. Lauder Brunton states that disease of the nose is liable to cause pain in the frontal region at the margin of the hairy scalp. Pain at, or a sensation of pressure on, the vertex is often complained of in hypochondriasis, hysteria, in women debilitated by prolonged lactation, anæmia, poverty, and about the climacteric. Pain at the back of the head is common in cerebellar tumour, in basal meningitis of children; it occurs also in chronic affections of the naso-pharynx. Von Tröltsch, while stating that disease of the naso-pharynx is a common cause of pain in any part of the head, noted that examination of that space with the finger often produced pain at the occiput. Occipital headache occurs in persons who have evidences of nervous breakdown, in constipation, and in rheumatism of the scalp and nucha. Tenderness on pressure and pain on tapping are common in rheumatism of the scalp; the hairy scalp is more liable to be tender than the parts without hair, the vertex more than the occiput. In organic disease pain on tapping or even on pressure over a limited area often indicates the seat of lesion—affection of periosteum or of bone, or a tumour.

TREATMENT.—The discovery of the cause in many cases indicates the cure. To enter with detail into the treatment of headache would involve the writing of a small treatise on the practice of physic, the number of causal conditions is so great. For the treatment of these the reader is referred to the various subjects—meningitis, anæmia, uræmia, etc. In some cases the first treatment must be palliative, but if in any case the only prescription is antipyrin and rest, it is a practical acknowledgment of failure of diagnosis. In more chronic cases the treatment is at once directed to the cause. For example, if the nose be at fault, the diseased condition must be treated *secundum artem;* if the eye be to blame, the error must be corrected by means of lenses, or prisms, or the necessary combinations of these. Carious teeth may require treatment, and if impacted teeth be the cause the removal of one or more may be necessary; it is often good practice to remove the first permanent molars if they show evidence of caries, in order to obtain room, and often in older persons the wisdom teeth must be extracted. Headache due to periostitis from caries may occasionally be cured by painting the neighbouring gum with equal parts of liquor iodi fortis and of linimentum aconiti; half a minute later rinse the mouth with tepid water, and repeat the painting if required. Occasionally we are called to treat severe headache in association with acute gastric catarrh; nothing gives more rapid relief than a sinapism to the epigastrium and a dose of castor oil. When constipation or hæmorrhoids are the cause these must have suitable treatment.

In every case of severe headache the patient should be put to rest, with all sources of peripheral irritation removed; ladies often find relief from loosing down the hair. In some fevers and inflammatory conditions the hair should be removed, and cold applied either by means of Leiter's coil or an ice-bag. The ice-bag should not be kept constantly applied, but intermittently; continuous application produces severe pain. Headache due to the congestion of frontal sinuses in catarrh and influenza is best relieved not by cold, but by the frequent application of very hot compresses to the forehead.

In simple *congestive* headache the patient should be put to rest with the head high, a mustard foot-bath, a mustard blister, or turpentine stupe to the nape of neck, a saline aperient, and if the heart be acting violently, small doses of tincture of aconite repeated frequently till relief.

Headache due to chronic congestion has occasionally been cured by free epistaxis, therefore local blood-letting by leeches may in such cases be tried. Occasionally a cantharides blister to the nape of neck has relieved when other measures have failed. Any heart condition causing passive congestion should be treated.

In headache due to *anæmia*, we have also to do with debility of nerve-centres from malnutrition, therefore fatigue and exertion are to be avoided, rest in the recumbent posture favours the free flow of blood to the brain. Arsenic and other treatment for anæmia.

Headache due to exhaustion from fatigue may be relieved by rest, and sipping hot tea, coffee, or soup. Small doses of strychnine or of nux vomica are also useful. If the cause be worry in a nervous individual, 30 grains of bromide of potash, with 4 or 5 minims of tincture of nux vomica, or a dose of bromo-soda (bromide of sodium with caffeine), or a tabloid of phenacetin 4 grains, and caffeine 1 grain, or antipyrin 10 grains. Any of these may be repeated an hour later, if required. Externally, liniment of chloroform alone, or with an equal part of liniment of aconite, may be applied to the painful area.

In nervous debility, in addition to the immediate treatment of headache, diminution of work is necessary; for school children it is often necessary to curtail the amount of work, specially of home preparation. In severe cases complete abstention from it for a long period may be required, change of air and scene, open-air exercise, nourishing diet—fish, egg, and milk. Fellow's syrup or Easton's syrup, with arsenic. Occasionally in such cases, with frequently recurring headache, 10 or 15 grains of bromide of sodium, with tincture of nux vomica, after meals is useful along with some other tonic. Fletcher's syrup of the hydrobromates, with strychnine, is also useful.

In loaded bowels, with liver derangement, a dose of calomel, followed by a purge, gives rapid relief. Some persons who live highly are accustomed to relieve their frequent headaches by blue pill, or calomel, followed in the morning by a saline; the removal of the cause would be better attained by the regulation of the diet and an increased amount of exercise. It is the experience of many medical men, that although mercurials are useful, they favour a recurrence of the morbid condition of liver.

In *uræmic* headache do not give digitalis, but a dose of calomel as a bowel antiseptic, followed by a saline, and at first only water as food. Mustard to the nape of the neck. For the use of blood-letting and subcutaneous transfusion of normal saline solution, see "Uræmia."

In *rheumatic* and *syphilitic* headache, the most useful drug is potassium iodide, 5 to 20 grains three times a day after food. In all cases of chronic headache, even if there be no history of syphilis and no evidence of it, iodide of potash should be tried. In rheumatic headache extract of colchicum, ½ to 1 grain at bedtime, is often effectual, and in rheumatism of the scalp, with tenderness to touch, ammonium chloride, 15 grains in extract of liquorice and water every four hours, often acts like a charm. In gouty headache both potassium iodide and colchicum are useful.

Migraine.—In a considerable proportion of cases of acute headache to which the medical man is called he will find that he has to do with migraine. His treatment must be (*a*) of the attack; (*b*) during the interval. (*a*) Of the attack. The majority of patients have most relief lying down, others must sit; irritation of end-organs is excluded as far as possible, the room is darkened. In mild attacks 30 to 60 grains of guarana powder in infusion gives relief (the tincture is also said to be good); others find that a drachm of bromo-soda cuts short the attack, some use antipyrin, others phenacetin and caffeine. Placing the hands and feet in hot water gives relief in some cases. Some compress the carotids. In some the only means of obtaining relief from the agonising pain is the subcutaneous use of morphine. Such a method should be avoided, if possible. (*b*) Between the attacks. As the nervous system is profoundly involved, endeavour to raise its tone, and avoid everything which might lower it. In some persons uric-acidæmia is a common exciting cause of the attack, therefore in these the diet should be carefully regulated. Haig advises that nitrogenous food should be reduced to a minimum. Some persons enjoy comparative freedom if they abstain from butcher's meat and take only fish. Lauder Brunton advises a nightly dose of sodium salicylate, 15 to 30 grains alone, or with potassium bromide, 10 to 30 grains. Should the salicylate disagree he gives potassium bicarbonate. In some cases the persistent use of ammonium chloride after meals gives freedom. Wilks strongly recommends a nightly dose of cannabis indica, ¼ to 1 grain.

Occasionally obstinate headache, which has resisted other remedies, has been cured by the administration of an anthelmintic. In countries in which lumbricoids are common, headache in children is treated with worm nuts; here santonin in castor oil is best. In other cases a dose of ethereal oil of male fern has caused the expulsion of tape-worm, and with it the cure of persistent headache. Sometimes headache has been cured in ladies by cutting the hair short.

In the treatment of chronic headaches many drugs have been used in addition to those already named, both nerve tonics and sedatives. Quinine, zinc, valerian, ergot, butyl chloral hydras, etc. Turpentine was highly praised by Warburton Begbie—20 drops repeated after an hour or two. Balthazar Foster found that nitrite of amyl relieved headache like magic. This remedy and nitro-glycerine might be expected to be useful in the spastic variety (hemicrania sympathetica tonica). Ergot has been recommended in congestive headache, and Campbell recommends that it be tried if other remedies fail. Several writers advise the use of electricity. Campbell states that it has not received the praise due to it because it is seldom used in temporary and occasional headaches, he has occasionally found the pain of intracranial disease temporarily removed by the constant current.

In all cases, specially chronic cases, we must remember that we have to treat a peculiar nervous system, that it should through hygienic and dietetic measures be braced up, that exercise in the open-air is important, that the cause of headache should, if possible, be discovered and removed, and that as rarely as possible should our treatment be only palliative. Alcoholics and morphine to be used as rarely as possible.

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Head-Shaking.

HEAD-SHAKING AND HEAD-NODDING WITH NYSTAGMUS IN INFANTS (SPASMUS NUTANS).

SYNONYMS.—Head-jerking ; Head-rotating ; Gyrospasm ; Nodding spasm.

Definition.—This is a functional co-ordination-neurosis affecting infants under two years—generally when between four and twelve months old.

Clinical Features.—Its two cardinal symptoms—involuntary nodding or shaking of the head and ocular nystagmus—are usually both present, but either may begin some weeks before the other. Sometimes, however, only head-movements are observed throughout ; while in other cases there is passing nystagmus only.

The movements of the head may consist in a simple forward nodding, but lateral or rotatory shaking is more common. They cease when the child is lying down, and also when the eyes are closed voluntarily or artificially. The nystagmus is rapid and of short range. It is oftenest horizontal, but may be rotatory or vertical ; in the latter case the upper eyelid may participate in the movement. It is generally more marked in one eye than in the other, and it is common to find one eye only affected. Occasionally there is rotatory or vertical nystagmus of one eye, and distinctly horizontal movements of the other. When the head is passively steadied by the hand the nystagmus increases ; or it may become visible then for the first time.

Rhythmical contraction and dilatation of the pupil (hippus) may sometimes be found, and occasionally convergent strabismus develops. The child has often a peculiar trick of turning his head to one side and staring fixedly out of the opposite corners of his eyes. This gives him, at times, a curious vacant preoccupied look. The intellect, however, is not at all affected however severe or long-continued the symptoms may be. When the child's attention is attracted in any way this seems to increase the movements. They diminish with drowsiness and cease during sleep.

Course and Duration.—The patients are generally weakly children—very often rickety—and in unsatisfactory hygienic surroundings. The symptoms usually begin suddenly, almost always in mid-winter. They seldom last less than six weeks, usually from three to six months, and sometimes longer. Occasionally, after complete recovery, there is a return during the following winter.

Etiology.—There are many factors contributing to the causation of this condition. Falls on the head and the irritation of teething have been blamed. The most important influences, however, in determining the onset of the disease are the age of the patients, the absence of sufficient sunlight in their surroundings, and the presence of rickets. It is also probable that anything else which temporarily or permanently lowers the vitality predisposes to its occurrence and to its long duration.

Diagnosis.—Cases of spasmus nutans are usually easy to recognise. Eclampsia nutans, or the "salaam convulsion," has sometimes been mistaken for it, but is quite different, being a form of epileptic seizure associated with serious cerebral defect. It is also not difficult to distinguish these cases from those in which somewhat similar head-movements are due to chronic hydrocephalus or to some other organic brain-disease, and in which the prognosis is naturally altogether less favourable. The nystagmus of these cases can often be distinguished from more serious forms by its having recently set in without obvious cause, and by the movements being confined to one eye or being different in the two eyes.

Treatment.—While sedatives may appreciably diminish the extent of the movements they are not of much importance in the treatment. The chief thing is to secure abundance of fresh air and sunshine, and to put the child on an antirachitic regimen with cod-liver oil and phosphorus.

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Health Resorts. *See* THERAPEUTICS.

Hearing, Artificial Aids to.

See also AUDITORY NERVE, EAR.

THE artificial aids to hearing may be divided into two groups, those which by their action improve the impaired function of the diseased ear, and those which, by collecting the waves of sound, bring them to bear upon the organ in a more concentrated form. To the former group belong the varieties of so-called "artificial tympana." The latter group comprises the various forms of ear-trumpets, conversation tubes, etc.

1. *Artificial Tympana.*—Field gives a nearly complete list of the different artificial ear-drums invented by surgeons. They are as follows:—1. Yearsley's cotton-pellet. 2. Turnbull's cotton-ball attached to thread. 3. Turnbull's disc of adhesive plaster. 4. Turnbull's disc of sublimated gauze. 5. Thomas' disc of oiled silk. 6. Barr's cotton-pellet fixed by collodion. 7. Blake's disc of sized paper. 8. Field's disc of india-rubber, cotton-wool, and flannel. 9. Burkhard Merian's solid disc of india-rubber. 10. Toynbee's solid piece of india-rubber on a silver wire stem. 11. Politzer's india-rubber tube as long as the meatus. 12. Lucæ's india-rubber disc attached to a rubber tube. 13. Downie's circular patch of egg pellicle. 14. F. H. Pierce's disc of stout linen with cotton thread soaked in vaseline. 15. Richardson's gold cylinder. 16. Michael's glycerine thickened with tannin. 17. Farquhar Matheson's plug of powdered boric acid. 18. Ward Cousin's hat-shaped membrane of compressed cotton.

To these may be added the most recent of all, Dadysett's combined trumpet and ear-drum.

Of these artificial tympana, Toynbee's instrument and all its modifications are to be condemned. They are the favourite pattern of the aural quack, and, at the meeting of the British Medical Association in 1888, the opinion was expressed that the so-called "ear-drums" largely advertised, and consisting of a piece of india-rubber fixed to a metallic stem (Toynbee's form), often cause serious injury to persons already suffering from defective hearing.

The variety most employed by otologists is the original cotton-pellet of James Yearsley, first introduced into practice in 1848. This simple contrivance owed its origin to the following occurrence:—In 1841 a gentleman from New York consulted Yearsley for large perforations of both membranes. On being told the condition of his ears, he said: "How is it, then, that by the most simple means I can produce in the left ear a degree of hearing quite sufficient for all ordinary purposes?" He did this by means of the "insertion of a spill of paper, previously moistened at its extremity with saliva, which he introduced to the bottom of the passage."

Yearsley's artificial tympanum consists of a small pellet of cotton-wool, either twisted up with a tail, or tied about its middle with a cotton thread. Hartmann prefers a pellet with an elongated tail, the latter tied round with thread, and the whole waxed.

The pellet probably acts as its inventor originally believed, viz. by supporting the ossicular chain. Yearsley believed that the membrane has an important action in supporting the ossicular chain, and that the pellet restored this support when lost by perforation. In some cases of old non-suppurative middle ear inflammation with a lax membrane the cotton-pellet will greatly improve the hearing power.

The advantages which may be claimed for Yearsley's cotton-pellet are:—(1) It is simple and effective. (2) The patient can make it for himself. (3) It can be made of antiseptic wool. (4) It is easy of manipulation. (5) It is well tolerated by patients. (One of Knapp's cases wore one for 29 years.)

The cases best suited are those in which the membrane is partially or totally lost, but it will be found that in all varieties of perforation, from small holes to almost complete loss of the membrane, there will be cases in which the application of the artificial drum is attended with marked benefit, while in others apparently similar no good can be obtained even after long perseverance. It is especially where there is a solution of continuity in the ossicular joints (particularly in that between the incus and the stapes) that the pellet will be of service.

When the malleus and incus are present the pellet requires to be placed in contact with them. In cases where the head of the stapes is free the wool must be placed against that bone itself.

The presence of profuse discharge is a contra-indication to its use.

Directions as to the application of the cotton-pellet have already been given and need not therefore be recapitulated.

2. *Ear-Trumpets, etc.*—A large variety of trumpets, conversation-tubes, and other instruments have been devised and introduced from time to time as aids to hearing. Amongst them the aural quack finds a happy hunting-ground, and advertises freely his cornets, auricles, acrophones, apparitores auris, and similar valueless arrangements to catch the money of the unwary. These quack instruments may be at once dismissed from consideration, they are almost always useless, many cause considerable irritation and even ulceration of the meatus, and the smaller ones may sometimes be found in contact with the membrana tympani bathed in the foul pus of a suppuration caused by their presence.

The special function of the auricle and meatus is the collection and reflection of the sound waves, whereby as few of them as possible are lost. The natural and fundamental form of artificial "ear-trumpet" is the placing of the hollowed hand to the ear. This augments resonance and increases the auditory capacity, especially for notes of high pitch and short wave-length, such as are found in the overtones of the human voice. All the trumpet forms of artificial aid are designed to act similarly. They are made of various substances—ebonite, iron, glass, porcelain, silver-plated metals, etc., the best reflectors being those of hard and dense material. Against their employment, however, their weight, fragility, and intrinsic notes greatly militate, and therefore such light metals as aluminium are often used. The simplest (and, therefore, the best) form is that of a hollow cone, and this, modified in various ways, is that usually adopted for trumpets. Echoes may be lessened by widening the cone-base and obliquely truncating it. Some years ago Dr. C. J. B. Williams published the result of certain experiments with these aids to hearing. He found that the confusion of transverse vibrations could be obviated by perforating the sides of the trumpet. He pointed out that a cone of stiff paper, some eighteen inches long, ending in a short metallic ear-piece, caused very little reverberatory roar, and magnified sound twelve times.

The modifications of these aids are so numerous that space will not allow of any complete description of them, and, indeed, such description would not serve any useful purpose. Otologists are practically unanimous in the opinion that, with the exception of conversation-tubes and simple forms of trumpet, these aids to hearing classed in this second group are useful in but a small proportion of cases.

A few words must be said regarding "audiphones" before discussing tubes and trumpets in more detail. The audiphone is a fan-shaped disc of vulcanized rubber, bent by a silken cord. The edge is placed in contact with the upper teeth, the convex surface being turned toward the source of sound. The waves of sound caught thereon are conveyed to the internal ear by bone-conduction, *via* the upper teeth. The contrivance will, however, be found of little value save in a few exceptional cases.

From what has been already said, it will be gathered that of this class of artificial aids to hearing only two forms are of actual practical value:—

(*a*) The conversation-tube.

(*b*) A simple form of trumpet.

(*a*) *The conversation-tube* consists of a trumpet-shaped mouthpiece, to collect the sound waves, connected with a metal or vulcanite earpiece by means of a piece of tubing of covered rubber strengthened by spiral wire. The mouthpiece is held by the person with whom communication is required, who speaks into it in ordinary tones. Here it may be remarked, that in conversing with deaf persons a distinct enunciation in ordinary tones will always be heard the best. The greater part of the sound in spoken words is produced by the vowels, and it is the consonants which form the distinguishing part of words and which have the softest tone. The louder one speaks, the more intense is the sound of the vowels as compared with the consonants; hence in shouted or loudly-spoken words the former are apt to eclipse the latter, and the distinctiveness of the words is thereby impaired or lost. Shouted conversation is therefore worse heard than that uttered in ordinary tones.

The conversation-tube can be worn round the neck or carried coiled up in the pocket, and is decidedly superior for near conversation.

(*b*) A simple form of *trumpet* is required for hearing at a distance; that which is, *par excellence*, the best is the one known as the *London horn*. It is made in three sizes, and should be painted a dull black, the plated ones being somewhat conspicuous. It consists of a bell-shaped receiver, from the side of the large end of which springs a curved tube, ending in an ivory, ebony, or vulcanite earpiece. The mouth is usually covered with a fenestrated disc. The earpiece is introduced into the meatus, the large open end being directed towards the source of sound. This instrument is excellent for churches, concerts, lectures, theatres, and the like. A noticeable drawback to the metal horns lies in the metallic adventitious sounds which they convey, particularly when listening to singing, orchestral music, etc.

Among other artificial aids to hearing may be mentioned the "rodostephone" of Thomas, and the modification thereof of Cresswell Baber. These are used after the manner of the "audiphone" already described, and aim at transmitting sound though the teeth and the bones of the skull. Their use, however, is very limited.

At one time much was expected of the microphone as an aid for the deaf, but hitherto all results from that invention or its modifications have proved impracticable and disappointing.

To sum up the subject, therefore, it may be said that of all the artificial aids to hearing that have hitherto been devised, only three are of any real practical value, namely, a simple and unirritating form of "artificial ear-drum," the conversation-tube, and the London horn.

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Heart.

GENERAL INDEX OF ARTICLES.

1. **Embryology, Comparative Anatomy, and Physiology of.**
2. **Affections of Myocardium and Endocardium.**
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4. **Congenital Affections of.**
5. **Surgery of.**

Heart—Embryology, Comparative Anatomy, and Physiology of.—It is necessary to the understanding of diseases, disorders, and malformations of the heart, that we should bear in mind the origin and development of the organ, as well as particulars in its comparative and essential anatomy and physiology. It is, therefore, proposed to state as clearly and as shortly as possible such points in the embryology, comparative anatomy, essential structure, and functions of the normal organ, as are calculated to explain its behaviour when diseased, disordered, or malformed.

THE EMBRYOLOGY OF THE MAMMALIAN HEART.—Of the three layers of the germ, the ectoderm, the mesoderm, and the endoderm, whence all parts of the organism are derived, it is the mesoderm which contributes chiefly to the formation of the heart. The endocardial lining of the organ is alone derived in all probability from the endoderm, which, however, as the "endothelial heart," is a very distinct structure in early fœtal life. The cerebro-spinal nerves, which ultimately penetrate and permeate the organ, are alone derived from the ectoderm, while recent research has confirmed the correctness of Remak's belief that the sympathetic ganglia spring from the mesoderm like the muscular, vascular, parenchymatous, and pericardial constituents of the heart (Paterson, *Phil. Trans. B*, London, 1890).

While these three sources are welded into the unity of a whole in this organ, as throughout the body, there is thus a largely preponderant community of origin between the muscle, vessels, sympathetic nerves, parenchyma, and pericardium of the heart.

The embryonic heart in the mammal is at first a bilateral tubal organ placed at the cephalic extremity of the medullary groove, which coalesces by fusion into a single tube, and finally emerges from development as the unified though bipartite heart of completed mammalian growth. At a very early stage the embryonic heart, which continues throughout essentially a single tube, acquires the property of rhythmical pulsation. As it grows lengthwise it accommodates itself to the somewhat confined space destined for it, by bending upon itself. Early in development also, the heart of the amniote embryo, whether human or other, not only exhibits this accommodative bending, but also shows variations in its diameter which indicate its division into auricle, ventricle, aortic bulb, and the channels which unite these portions. The earliest indication of the cardiac apex is given by the bending and close apposition of the ventricular portion of the tube, best seen in the inner or endothelial heart, while as yet the auricular and aortic portions are at some distance from the ventricular, and separated from it by the auricular canal and so-called fretum Halleri. These connecting channels are the sites of the future valves. With this continued growth of the organ the auricular portion assumes a bipartite configuration, and of this portion of the still single chamber the right division is the larger. A similar indication of division in the ventricular portion is now seen, and of these still communicating divisions the left is the larger. At this stage the aortic bulb turns towards the middle line and upwards, so that the rest of the aorta proper comes to lie between the two auricles.

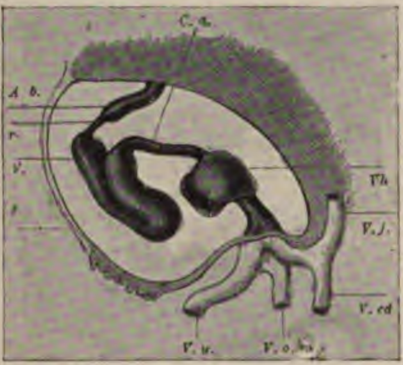
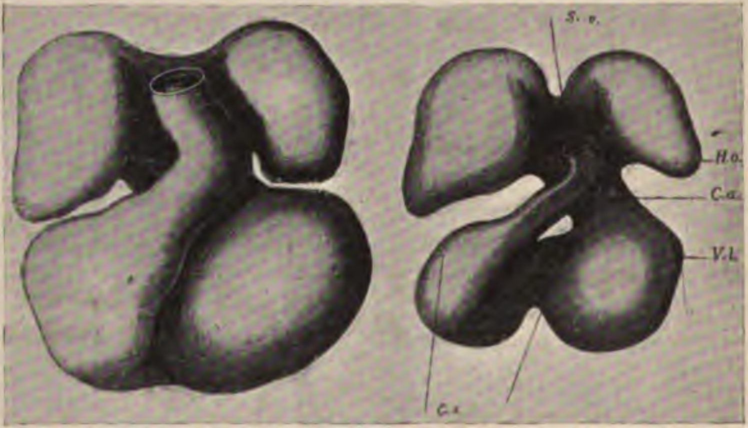


FIG. 1.—The endothelial heart seen in profile (His). *A.b.* aortic bulb; *r*, fretum Halleri; *v*, ventricle; *C.a.* auricular canal; *V.j.* jugular vein; *V.cd.* cardinal vein; *V.o.m* omphalo-meseraic vein; *V.u.* umbilical vein.

With these variations in external configuration certain internal changes are gradually associated. Septa arise from the roof and floor of the auricular portion, which ultimately completely divide this part into two distinct chambers. A septum likewise springs from the floor of the ventricular portion, which finally erects a complete partition and brings into being two distinct ventricles. With this septal division, a valvular demarcation has gradually associated itself, and the chambers of the once common cavity are shut off, not merely auricle from auricle and ventricle from ventricle, but the corresponding chambers to the right and left from each other by means of the auriculo-ventricular valves.

While the heart is still a comparatively undifferentiated tube, its aortic bulbar end is in connection with a bilateral system of five aortic arches, the obliteration of some portions of which and the persistence of others finally constitutes the adult mammalian arterial system. The obliteration of the right and left first and second arches, and of a portion of the stem between the third and fourth, leaves the permanent channels of the internal and external carotid arteries; while the obliteration of the whole of the right fifth and of the stem connecting it with the right descending aorta, leaves the fourth right arch to constitute the right subclavian artery and its branches. The fourth left arch remains as the permanent transverse and descending aorta, while the fifth left arch forms the ultimately obliterated *ductus arteriosus.* The persistent stems between the right and left third and fourth arches remain as the common arteries. From a septation and division of the aortic bulb the pulmonary arteries come into being.



FIGS. 2 and 3.—Muscular and endothelial heart of the embryo (His). *S.v.* sinus vestibuli; *H.o.* auricle; *C.a.* auricular canal; *V.l.* left ventricle; *C.s.* conus arteriosus.

At the caudal end of the heart certain changes occur in the embryonic venous system, which result in the persistent veins. In the pre-placental embryo venous blood is collected from the vascular area, and brought into the body by two large vitelline or omphalo-meseraic veins, whose destiny is to become the portal venous system of the adult. With the appearance of the placenta blood is brought from that important organ by two umbilical veins ultimately fused into one in the cord, but persistent for a time thereafter as two in the abdominal cavity. The umbilical and vitelline veins, together with the duct of Cuvier—a trunk which collects the blood from the upper or jugular section of the primitive venous system and the lower or cardinal—open into the right and left venous sinuses which are continuous with the auricular heart.

These sinuses ultimately coalesce, and the dextral entrances constitute the inlets of the permanent systemic venous system, while the pulmonary veins, after the formation of the lungs, enter the sinistral portion of the sinus.

We have seen that in the formation of the ultimate arterial system a larger portion of the right than of the left aortic arches is obliterated in the course of development. In the early bilateral venous system the reverse is the case. In the establishment of the persistent veins a larger portion of the left than of the right primitive system is obliterated, except in rare and exceptional instances. The arteries, like the projected arterial stream, grow into the body; the veins, like the returning venous current, grow in a measure out of it. Thus, with the formation of an obliquely transverse communication between the upper part of the left primitive jugular vein and the lower part of the right corresponding vessel, the current from the lower portion of the vessel on the left is in great measure diverted from it, and the channel shrinks finally to obliteration, with the exception of a portion at its lower end which persists as the coronary sinus, and a part at the upper end which remains as the superior intercostal vein. The transverse branch, and the cause of this diversion of current, becomes the left innominate vein, the future important recipient of the chief lymph stream from the thoracic duct. The relation of the thoracic duct and vein is of practical moment in retrograde stasis due to heart disease.

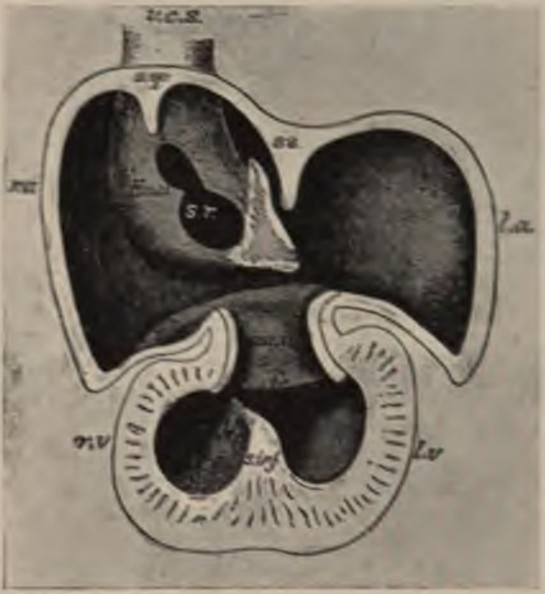


FIG. 4.—Septatim of the heart of the human embryo (Quain after His). *V.c.s.* Vena cava superior; *s.sp.* septum spurium; *s.s.* septum superior; *Eu.v.* Eustachian valve; *s.v.* sinus venosus; *r.a.* right auricle; *l.a.* left auricle; *au.c.* auricular canal; *s.inf.* septum inferior; *r.v.* right ventricle; *l.v.* left ventricle.

A similar but reversed process meanwhile takes place between the left and right cardinal veins. An obliquely transverse communication is established between the lower portion of the left and the upper part of the right cardinal vein, and persists as the vena azygos minor. It coalesces with the right cardinal vein to constitute the larger azygos vein. The confluence of the left and right innominate veins thus constitutes the mighty stream of the superior vena cava, which is further swollen before it debouches into the right auricle by receiving the vena azygos major. The veins from both sides of the body have meanwhile coalesced more or less in the middle line to form the inferior vena cava.

The important remaining and intervening portion of the venous system is completed, as has been stated, by the changes which take place in the development of the vitelline or omphalo-mesenteric and the umbilical veins, which result in the formation of the portal venous system and its supra-hepatic outflow the great hepatic vein.

The great hepatic and umbilical veins are further united during later fœtal life through the medium of the ductus venosus, which, like its arterial analogue, the ductus arteriosus, disappears after birth. The disappearance of these channels coincidently with birth is, however, due to opposite causes. The ductus arteriosus disappears, because the breathing organ of adult life—the lung—has come into action; the ductus venosus vanishes because the breathing organ of fœtal life—the placenta—has ceased to exist.

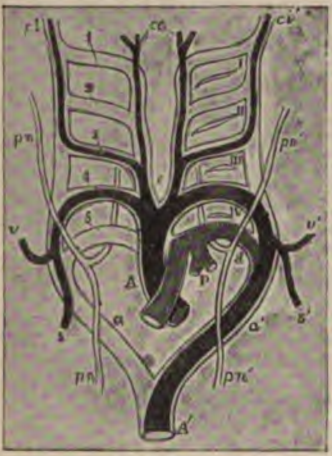
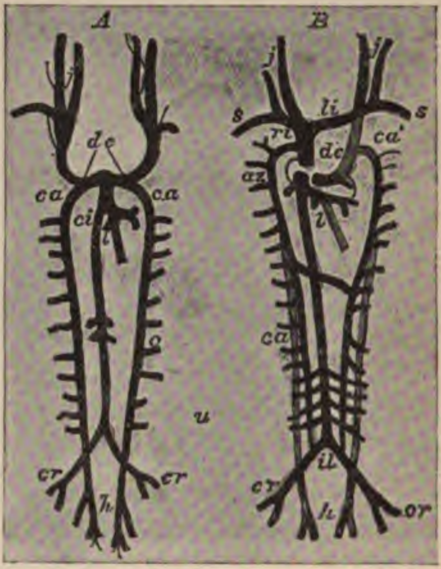


FIG. 5.—Diagram of the transformation of the aortic arches into permanent arteries (Quain after Rathke). A, P, ascending aorta and pulmonary arteries; 1, 2, 3, 4, 5, right branchial arterial arches; I, II, III, IV, left branchial clefts with arches corresponding to those on the right; *ci*, *ci'*, right and left internal carotid arteries; *ce*, external carotid arteries; *c*, common carotid arteries; *v*, *v'*, vertebral arteries; *s*, *s'*, subclavian arteries; *d*, ductus arteriosus; *a*, *a'*, right and left aortic roots; A', permanent descending aorta; *pn*, *pn'*, right and left pneumogastric nerves. Obliterated portions left unshaded.



FIGS. 6 and 7.—Diagram of the condition of the great veins in the embryo, and of their transformation into the permanent veins (Quain after Kölliker). *j*, Jugular veins uniting with the subclavius; *s*, *ca*, cardinal veins becoming the azygos as in B; *dc*, ducts of Cuvier formed by the union in BA of jugular and cardinal and becoming the superior vena cava on the right side, but disappearing on the left side; *ca'*, part of left cardinal vein which disappears; *ci*, vena cava inferior; *l*, hepatic veins and ductus venosus; *il*, common iliac veins; *cr*, external iliacs; *h*, hypogastric becoming the internal iliacs. Portions which disappear less darkly shaded than the permanent portions.

Notwithstanding the confluence of the two great venous channels, the upper and lower venæ cavæ at the right auricle, there is, during the greater part of fœtal life, little admixture of the two streams. The difference in direction of these, the large development of the Eustachian valve at the entrance to the inferior cava, together with the direction given by the auricular septum and its deficiency at the foramen ovale, serve to direct the inferior blood-stream—that most oxygenated by placental respiration—across the right into the left auricle, and thence by way of the left ventricle and the arteries of the aortic arch into the upper part of the body; while the blood returning by the superior cava to the heart, and therefore the less oxygenated, passes through the right auricle into the right ventricle, and is sent thence chiefly by way of the ductus arteriosus into the abdominal aorta and the territory irrigated by the latter. The situation of the ductus arteriosus or Botalli and its entrance into the aorta beyond the primary vessels of the aortic arch is a beautiful provision of Nature for supplying the most nutrient fluid to the organs most necessary to fœtal life, namely, the heart, by way of the coronary arteries and the growing nerve-centres indispensable to organic life.

While a portion of the pulmonary circulation is probably pervious *in utero*, and circulates the less oxygenated blood contained in the right ventricle, the effect of the respiratory act at birth is to open out the whole of the pulmonary blood-vessels, and thus to accommodate a large quantity of blood. Pressure in the left auricle is thus suddenly raised, the foramen ovale is closed by its valve-like screen, a change in the position of the heart is brought about, which, according to Schantz (Pflüger's *Archiv f. d. gesammte Phys.* Nov. 1888), twists to some extent the ductus arteriosus, and the circulation then assumes the character of that of adult life. The fœtal channels are, as a rule, altogether obliterated within a period variously estimated from a week to ten days.

THE COMPARATIVE ANATOMY OF THE MAMMALIAN HEART.—Professor Huxley has shown (*Man's Place in Nature*, Edin. and Lond. 1864) how the human fœtus in its development is an epitome of the process in vertebrates lower than man in the scale of being. He also suggests that the unity of the process may extend to still lower creatures. Support is found to the truth of this contention by a comparison of the development and anatomy of the heart of the higher vertebrates already given, with the characters and conditions of the heart in the non-vertebrate and lower vertebrate animals.

In the lowest organisms which take in solid food the nutritive material is mainly distributed from the digestive spaces directly into the protoplasm of the body (*Elements of Comparative Anatomy*, by Carl Gegenbaur, translated by F. Jeffrey Bell, p. 50, 1878). The next step in differentiation is the existence of a distinct digestive tube, through the walls of which nutriment passes into the body protoplasm. A little higher and we find the mid-substance or mesoderm of the organism containing a cavity—the cœlum—distinct both from its digestive tube and outer layer or ectoderm. The cœlum contains a fluid which changes its position during alterations in the configuration of the organism as a whole. Gegenbaur suggests that this is to be regarded as the first indication of a circulating nutritive fluid (*loc. cit.*). Later, canalicular cavities regularly arranged form the prototype of a vascular system, which a little later still open into a perienteric space, and we then reach the confines of the completely shut off heart and vascular system of vertebrates. This progress in development is well shown by a series of vascular apparatus figured by Gegenbaur to illustrate the circulation in Mollusca (*op. cit.* p. 368). These show the transition from a rhythmically contracting tube sending its contents anteriorly and posteriorly, to one receiving fluid posteriorly and transmitting it anteriorly as in the mammalian heart, the receiving cavities being the prototypes of the auricles, the transmitting of the ventricles. Between these two divisions and distinguished by the direction given to the circulating fluid, there is the remarkable phenomenon evinced by the Tunicata of a rhythmical vascular system propelling its contents for a time entirely in one, and then after a short pause by a process of reversed rhythmicality in the opposite direction (Gegenbaur, *op. cit.* p. 406).

It is, however, the persistent characteristics of the heart in the craniote vertebrates which have most interest for students of the circulation and its disorders in man, inasmuch as some of these offer us types of those malformations which occur in the fœtal heart from arrest in development of, or occlusions taking place in portions of the heart, during the various phases of its growth.

The determining factor in the development of the heart in the Craniota appears to be the situation of the organs in which blood already used by the body is aerated for further use. When such organs, as in the case of fishes the gills, are, so to speak, outside the body, that is, directly exposed to external aerating conditions, it is manifest that the oxygenated blood returned to the body requires only a single-chamber system for its reception and propulsion within the body. We find, therefore, in fishes, that the heart consists of a single auricle and ventricle. In the amphibia, the characters of the heart in fishes persists to the same extent as the subaqueous habits of the amphibian. The nearer members of this class are to the fish in habits the more do their hearts resemble that of the fish. That is, although the auricle is divided more or less by bands, the two auricles, if they can be so regarded, still communicate freely, and in addition the ventricle is even less divided. It is, indeed, practically one chamber. The efferent tube is still an aortic or arterial bulb, a branchial system of arteries still exists, as well as pulmonary arteries, and, finally, a ductus arteriosus establishes communication between the pulmonary artery and aorta, as in the case of the mammalian fœtus.

The heart of the reptile shows a farther step in differentiation. The auricles are distinct, as also in great measure are the ventricles. In the crocodile the ventricular partition is complete. In those members of the group in which this separation is not perfect, as in the snake and the turtle, the communicating deficiency at the base of the ventricular septum is so obstructed by a strong muscular valve-like structure as to leave arterial blood in the left and venous blood in the right chambers of the heart. The blood is, moreover, directed by this arrangement, the arterial into the systemic arteries, and the venous into the pulmonary vessels. In some persistent malformations of the mammalian heart the reptilian heart is well represented. In birds, as in mammals, which exist chiefly in air (for the diving bird and the diving mammal (whale) must rise to breathe) the auricles and ventricles are completely divided, and the chambers are more capacious than in creatures lower in the scale of being. The difference between birds and mammals is rather in the character of one of the valves than in the chambers of the heart. The right auriculo-ventricular orifice in the bird is guarded by a strong muscular falciform fold or curtain. This is attached to the anterior wall of the right ventricle, and follows pretty accurately the curve of the conus arteriosus. The right ventricle as a whole is small, and is wrapped round the large and powerful left ventricle. The physiological significance of these conditions is interesting, and probably related to the regulation of the venous supply to the lungs under the very varying conditions of pulmonary inflation in flight and in repose.

Many of these conditions in the lower animals are, as we have seen, represented, during one period or another of development, in the higher mammals, including man. The external respiratory organ of the mammalian fœtus, its gills so to speak, is the placenta, and the corresponding cardiac condition is but a more complex form of the simple single-chamber system of the fish, which gradually increases in complexity to that of the more differentiated reptile, and ultimately reaches the perfect division seen in the heart of the adult air-breathing animal. In no process, indeed, is seen more perfectly than in the development of the mammalian heart the evidence of what the older writers called "design in nature," and it is difficult to use any other term even now. The adaptation up to a certain point of existent organs to present circumstances is a well-recognised fact in vital processes, whether physiological or pathological; but the preparation and perpetuation of organs and organisms for future use, and to meet conditions foreseen or intended, but not yet present, appears to some to be beyond the power of a mere acquired habit of growth or haphazard fusion of species. The controversy is, after all, however, but a war of words, for the results of "natural selection" are quite the same as those of appropriate combination, and the determination of causes from effects opens up a chain of reasoning one end of which, at least, is lost in infinity, and cannot be wholly explained by physiology alone. (For particulars concerning the anatomical situation of the heart, the reader is referred to the section on Clinical Investigation of the Chest, vol. ii. p. 200.)

The Physiology of the Mammalian Heart.—If the heart of a mammal be dissected after having been boiled sufficiently, it will be found that the arrangement of its muscular fibres has an important relation to its action as a vital organ. In the first place, the auricular portion of the heart may be detached from the ventricular, and the dividing line between these, which occupies the place of the auricular canal and fretum Halleri in the fœtus, is seen to consist of fibrous material of a more or less resistant character, which surrounds in great measure the auriculo-ventricular and arterial orifices of the heart, and enters into the constitution of its valves, tendons, and interstitial tissue. In some large animals, indeed, a portion of this dividing line situated between the aortic and the two auriculo-ventricular orifices becomes fibro-cartilaginous and even bony. In the next place, the arrangement of the muscular fibres of both auricles and ventricles shows that the organ in its growth has been at first bilateral and then unified. That is, there are fibres which seem to appertain to each half of the auricular and ventricular portions of the heart, and others which embrace both the right and left auricles or ventricles as the case may be. In the third place, the muscular fibres of the external and middle portions of the ventricular wall appear to be continuous with those which pass into the ventricular septum and papillary muscles (Pettigrew, *Phil. Trans.* 1864; Quain's *Anatomy*, vol. i.).

There is also some muscularity of the cardiac ends of the two venæ cavæ and of the pulmonary veins, which in quadrupeds at any rate is most marked in the case of the superior cava. Muscular elements are likewise said to enter into the constitution of the auriculo-ventricular valves (Foster).

Cardiac muscular fibre is of the striped variety, but the striæ are not so distinct as in skeletal muscle, and suggest a transition condition between the latter and unstriped visceral fibres.

The important physiological consequence of these arrangements is, that separate and consecutive action of the auricles and ventricles is secure, and that all parts of these chambers act simultaneously during the period of their respective contraction, and relax at the same time during their quiescence. As a succinct description of the cardiac cycle nothing can be added to the following lucid statement:—

"When the chest of a mammal is opened and artificial respiration kept up, the heart may be watched beating. The great veins, inferior and superior venæ cavæ and pulmonary veins, are seen, while full of blood, to contract in the neighbourhood of the heart: the contraction runs in a peristaltic wave towards the auricles, increasing in intensity as it goes. Arrived at the auricles, which are then full of blood, the wave suddenly spreads at a rate too rapid to be fairly judged by the eye, over the whole of these organs, which contract with a sudden sharp systole. In the systole the walls of the auricles press towards the auriculo-ventricular orifices, and the auricular appendages are drawn inwards, becoming smaller and paler. During the auricular systole the ventricles may be seen to become turgid. Then follows, as it were immediately, the ventricular systole, during which the ventricles become more conical. Held between the fingers they are felt to become tense and hard. As the systole progresses the aorta and pulmonary arteries expand and elongate, the apex is tilted slightly upwards, and the heart twists somewhat on its long axis, moving from the left and behind towards the front and right, so that more of the left ventricle becomes displayed. As the systole gives way to the succeeding diastole the ventricles resume their previous form and position, the aorta and pulmonary artery shrink and shorten, the heart turns back towards the left, and thus the cycle is completed" (Foster, *Text-Book of Physiology*, Part i. p. 232, London, 1893).

The length of time during which these events occur is estimated by Foster to be about 0·8 seconds, of which he apportions 0·3 seconds to ventricular systole, and 0·5 seconds to complete ventricular diastole which includes 0·1 second for auricular systole. In these calculations the heart is assumed to maintain an average rate of 72 beats to the minute (*op. cit.* p. 264).

With these movements of the heart two sounds and two pauses are associated. The first, a long, booming sound, occupies a considerable portion of ventricular systole, and is most intensely heard near its commencement; the second, a short, sharp sound, occurs during a small portion of ventricular diastole at its commencement. Between the first and second sounds there is a short pause difficult of measurement, and between the second and following first sound a considerable and more easily appreciable pause not audibly broken by the systole of the auricles. These facts may be represented in the diagrammatic manner first employed by Sir William Gairdner of Glasgow, and which has been variously modified by subsequent writers.

The explanation of pauses in action is of course easy. Absence of action is of necessity as noiseless as death itself. The short pause ensues when ventricular systole is spent. It is virtually the commencement of ventricular diastole, which is only broken by a brief passive event,—the closure of the semilunar valves, which emit a short, sharp sound on closing under the influence of blood-pressure in the aorta and pulmonary artery. The cause of the first sound has been much debated, and an examination of the method of its production has a practical interest. It may be heard when the thorax is opened artificially, and also when the heart is exposed by congenital defect in the chest wall. It cannot, therefore, be due to the heart being contained in the thorax, or to the impact of its apex against the chest wall. The events accompanying it are: (1) systole of the ventricles; (2) opening of the semilunar and closing of the mitral and tricuspid valves; (3) the pressure of blood upon all the constituents of the chamber which contains it; (4) the rise of this pressure over that of the column of blood contained in the efferent arteries, and (5) the consequent passage of blood into these. Foster (*op. cit.* p. 240), without entering into details, states that if the ventricular portion of the heart be cut off below the level of the auriculo-ventricular valves, the detached portion may be heard to emit sound. If this be so, it is highly suggestive of muscular contraction playing a part in the production of the sound, but that part may be a very small one. As regards the obscuration of a sound or sounds of the heart by defect in its valvular apparatus, careful clinical observation in many cases and in all varieties of valvular disease has convinced the writer that such obscuration is in direct proportion to the amount of noise generated and not to the extent of the lesion present in any particular valve. He has also known the heart-sounds to be clearly audible without murmur when both aortic and mitral valves were much diseased and the heart beating very feebly at a rate of 150 in the minute. The clear sounds probably arose in this case in the right ventricle, which was ascertained to be sound at the necropsy. In determining experimentally the rôle of the valves in the production of the first sound, not only one but all four would have to be placed out of action during cardiac systole. This has never been done so far as the writer is aware.

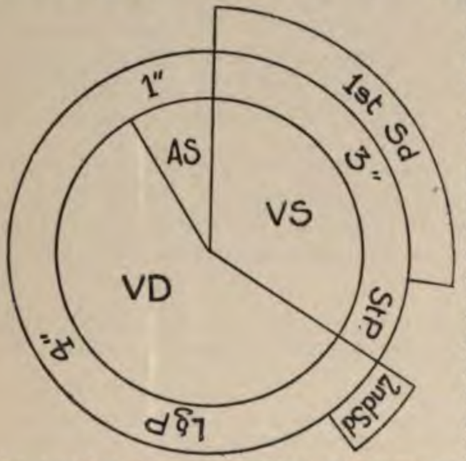


FIG. 8.—A.S., auricular systole; V.S., ventricular systole; V.D., ventricular diastole; 1″, 3″, 4″, notation of time occupied by phases in cardiac cycle; St.P., short pause; Lg.P., long pause; 1st Sd., first sound; 2nd Sd., second sound.

Although Dr. Halford (*The Action and Sounds of the Heart*, p. 25, 1860) believed that the first sound of the heart was due to tension and vibration of the auriculo-ventricular valves during ventricular systole, he proved on several occasions, by careful experiment before capable and credible witnesses, that when the systemic venous inflow was prevented by forceps, and in addition the flow from the pulmonary veins by grasping them between the fingers, sound ceased to be emitted during systole, and returned immediately blood was again allowed to enter the heart. Drs. Yeo and Barret (*Journal of Physiology*, vol. vi. p. 145) repeated Halford's experiment, but without grasping the pulmonary veins at the root of the lungs. They found that under these circumstances cardiac systole was still accompanied by some, but greatly diminished sound. Dr. Arthur Leared argued and endeavoured to prove that the cause of sound during systole was not in the apparatus of circulation, but in the blood itself, and believed that the impact of the moving ventricular blood against the more stationary aortic blood was the essential cause of the first sound of the heart (*Essay on the Sounds caused by the Circulation of the Blood*, London, 1861). Finally, Sir Richard Quain (*Proc. of the Royal Soc.* June 1897) expressed the opinion that the first sound of the heart was the result of the ventricular blood being projected by a spiral movement, as shown by Professor Pettigrew, against the aortic cusps held down by the aortic column of blood. This view he had held and expressed in 1852, but did not publish till recently. The difference between it and Dr. Leared's is only that Sir Richard Quain attached some importance to the intervening aortic valves as a factor. In performing the experiments on which in part his conclusions were based, he used an apparatus constructed out of a sheep's heart, in which the right chambers were cut away, the coronary arteries ligatured, and the mitral valves destroyed. A column of water was then made to press upon the aortic valves and the ventricle was also filled with water. When now systole and diastole were imitated by alternate compression and relaxation of the ventricle by the hand, a very good imitation of both sounds could be elicited. He was good enough to ask the writer's assistance in the performance of this simple experiment, and the latter can add his testimony to Sir Richard Quain's as regards the facts.

Taking all these data into consideration, it appears legitimate to conclude, that more than one factor enters into the production of the first sound, and that the chief place is to be assigned to the resistance offered by aortic blood-pressure to the ventricular blood-pressure when the latter is suddenly and forcibly rising above it. If this conclusion be accepted, it follows that the audibility and character of the first sound may, *ceteris paribus*, afford some evidence of the vigour of ventricular contraction.

The Coronary Circulation.—The nourishment of the mammalian heart by blood is chiefly effected by the coronary arteries, which spring from the aorta behind its two anterior semilunar cusps. The pulse in all arteries is systolic in time, and the coronary arteries are no exception to the rule. There is reason, however, to believe that the repletion of the aorta on cardiac diastole also imparts additional impulse to the coronary arterial circulation, while the strong valve in the coronary sinus prevents regurgitation from the right auricle. Sibson (*Medical Anatomy*, p. 73) observed that both arteries and veins on the surface of the heart became turgid and tortuous during systole, and again straight during diastole. This fact appears to indicate that when ventricular contraction is at its height, some pressure is exercised upon the smaller arteries and upon the venous outflow from the coronary sinus. The anatomical situation of the coronary vessels in the texture of the heart is admirably calculated, however, to reduce injurious compression or obstruction to a minimum, as may be seen from the following illustration.

Anastomosis between the two coronary arteries has been disputed by some anatomists, but appears to have been proved to exist by Dr. Samuel West (*Lancet*, June 1883, p. 945) by injected specimens. It is probable, however, that it is not very free, a circumstance for which the original bilaterality of the organ in embryo may in a measure account. The most important and most constantly active organ in the body thus receives a constant supply of recently oxygenated blood, a circumstance which, when associated with an otherwise normal constitution of the blood, appears to be a leading factor in the maintenance of a healthy state and normal action of the cardiac muscle.

We have now briefly examined the muscularity and vascularity of the heart, and the chief physiological questions of practical interest associated with these factors. It remains to examine shortly the third factor in the production of the functional unity of the heart's action.

The Cardiac Nervous System.—The nerves of the mammalian heart spring from the trunk of the vagus and from the inferior cervical ganglion of the sympathetic chain. The trunk of the vagus, however, is a mixed one, and contains fibres of the spinal accessory nerve which avoid the jugular ganglion on the vagus, and passing through its lower ganglion or "ganglion of the trunk," course to the heart in the vagus. These are said to be of small calibre like all efferent visceral spinal nerves according to Gaskell (*Journal of Physiology*, vol. vii.). The sympathetic spinal nerves of the heart are derived from the upper dorsal spinal nerves from the second to the fourth and perhaps the fifth, but chiefly from the second and third (see Plate, p. 442). The gathering point for these in the thoracic chain is the stellate or first dorsal ganglion, issuing whence they encircle the subclavian artery as the so-called ring of Vieussens, and thence by way of the lower cervical ganglion pass to the heart. Both vagal and sympathetic branches from either side having reached the organ contribute to the superficial and deep cardiac plexuses. Here in all probability an incomplete peripheral decussation occurs, so that the nerves from either side supply both halves of the heart or both aspects of the unified tube of which the heart consists. This seems to be proved by the fact that unilateral section of the nerves does not materially disturb the heart's action, while bilateral section soon proves fatal. The majority of the medullated fibres of the vagus lose their medulla in the cardiac plexuses, the majority of the medullated fibres of the spinal cardiac nerves in the ganglia of the sympathetic chain which intervene between their point of issue from the cord and their exit from the inferior cervical ganglion. For our anatomical knowledge of these important facts we are largely indebted to Gaskell of Cambridge and his successors in this country and abroad. Kölliker of Würzburg affirms his belief (*Gewebelehre*, p. 858) that nerves which retain their medulla in the visceral periphery are afferent and therefore sensory, as on their way from the cord or medulla they pass through or over intervening ganglion without being broken up by them. On the visceral distribution of efferent branches of both the vagal and sympathetic series ganglion cells occur, and as the function of these two streams differs, their ganglion cells must remain essentially as distinct as the nerves themselves, however agglomerated. The final distribution of the nerves to the visceral muscle is by fine nucleated plexuses, which end upon muscle cells both cardiac and vascular. In the opinion of the majority of present-day histologists they do this by so-called free ends, the existence of which a minority still disputes or considers doubtful. Such an arrangement, however, would seem to be necessary to the exercise of the separate functions of the different nerves. The muscle cell appears to be the term common to both, and the probable medium of interchange between them, by processes too little known for fruitful discussion at present.

By means of this mechanism the regulated action and nervous nutrition or trophation (to coin a convenient but not very euphonious term) of the heart is secured. Although the embryonic heart has a rhythmical action before it is supplied either with organised vessels or with nerves, the conditions of sustained rhythmicality seem to require an integrity of all three factors—the muscle cell, the blood which bathes it, and the nerves which regulate it, and in some obscure way exercise a trophic influence upon it. By means of the nervous system the heart may be retarded or even arrested in action, that is, inhibited; quickened in rate and in force or augmented; and weakened, or depressed. The channel for inhibition, depression, and trophation is the vagus; for augmentation the sympathetic spinal nerves. The spinal accessory fibres already mentioned are now regarded as the inhibitory fibres, and are efferent in action *towards* the organ. The depressor fibres exercise their influence *from* the heart by way of both ganglia on the vagus at the vaso-motor centres in the medulla, whence they induce a fall in the peripheral blood-pressure. They are therefore afferent or sensory in character.

The activity of trophic nerves, of which we are justified in assuming the existence, is probably efferent. Sensory and motor fibres likewise exist in the sympathetic spinal nerves and constitute the peripheral mechanism of augmentation. They are, indeed, believed by some to be the chief seat of cardiac sensibility, and the character and distribution of radiated pain in sensory disorders of the heart supports this view. The general effect of retardation of the heart's action is regarded as conservative of energy or anabolic; that of acceleration or augmentation as expending energy and leading to exhaustion or catabolism. It has been maintained by some that the existence of efferent ganglia on the trunks of visceral nerves, and the interposition of the sympathetic system between the cerebro-spinal centres and the viscera, may be the cause of the differences between somatic and splanchnic nervous action, whether affecting the heart or other involuntary organs. The whole subject is, however, far from clear at present, but a good deal has been done to elucidate it during the last ten years or so. Like the unification of the heart itself, the unity of the cerebro-spinal and sympathetic systems has been secured by a process of fusion, for there is good evidence to show that in their essence they are embryologically distinct, and spring from different layers of the germ, as has been stated at the commencement of this article. Although, finally, the sympathetic system is now considered to be wholly or almost wholly dominated in function by its cerebro-spinal connection, the question of a retention by it of a measure of efferent autonomy cannot yet be regarded as settled.

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Heart, Affections of Myocardium and Endocardium.

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See also PERICARDIUM, SCHOTT TREATMENT.

THE first part of this article consists of a consideration of all the various etiological factors, pathological processes, and their cardiac and systemic effects, followed by a description of the recognised types of heart disease.

Secondly: The symptoms produced by heart-failure irrespective of its special cause are considered, and certain peculiarities associated with special forms of disease are referred to.

Thirdly: The physical signs of the different varieties of heart disease are described in detail, and their common combinations according to the pathological state that gives them origin are sketched.

Fourthly: The treatment—hygienic, dietetic, and medicinal—of heart disease is considered.

It is evident that a pathological knowledge should precede the study of symptomatology, physical examination, and the principles of treatment, inasmuch as these last three can only be profitably studied in the light of pathological experience. As regards their *symptomatology* we are entitled to consider nearly all the forms of heart disease together, seeing that they all tend to, and generally eventuate in, that condition we term *venous stasis*, in which the blood accumulates, as it were, on the venous side of the circulation, giving rise to the ordinary symptoms to be described, while stasis of blood in the chambers of the heart is the common cause of the embolic process and its clinical manifestations. If, as physicians, we are ever to keep before us the "tendency to death" in the case we are treating, then in heart disease the expression of that "tendency" is *venous stasis*.

It is by *physical examination* and the signs thereby elicited that *differentiation* of the various forms of heart disease is accomplished.

But as regards treatment, inasmuch as it is to the heart muscle practically alone that our efforts are directed, it is allowable again to generalise, and to consider together all varieties of heart disease.

The pathological considerations that precede symptomatology, etc., will amply demonstrate the importance of obtaining in every heart case an accurate account of the patient's previous health, for without this it is impossible to interpret aright either the symptoms or the signs, to forecast the probable course that will be run, and to treat the patient with the best prospect of success.

A. GENERAL PATHOLOGY

I. ÆTIOLOGY

1. Exciting Causes.—These are best considered according to the *nature* of the excitant.

(1) *Mechanical.*—The cardio-vascular apparatus has long been viewed as a machine, and cardiac physics have very rightly become a recognised branch of physiological study. But while it is necessary to avoid the fettering influences of a mere mechanical conception, the action of physical agencies must be admitted as producing or inducing temporary derangements or establishing permanent defects.

Occasionally excessive exercise or unwonted exertion in a subject absolutely or virtually sound seems to act on the cardiac muscle or the aortic segments as an acute strain. Even trained athletes sometimes after exceptional physical effort have manifested evidences of cardiac derangement. Not a few cases are on record where in seemingly healthy adults after some unusual effort physical examination has clearly demonstrated a condition of cardiac dilatation. Prolonged exercise, such as is illustrated by some athletic sports, is peculiarly liable to exert strain on the right ventricle, but it must be admitted that such most usually occurs in the untrained or those constitutionally untrainable and unfit for athletic pursuits. The effects of excessive physical effort or ill-regulated drill on young recruits, and the consequences of forced marches even on seasoned men, is well known, and "soldier's heart" has become a well-defined clinical entity. As might be expected, the effects of mechanical influences are most frequently met with in men, and particularly during the years of greatest activity and physical vigour.

The consequences of violent effort are to be clearly distinguished from those of chronic strain. Many laborious occupations necessitate the maintenance of a state of persistent or long-continued exertion. Porters, navvies, miners, blacksmiths, workers in iron and other heavy metals, and like laborious occupations, are all liable to cardiac derangement and failure from the unavoidable strain associated with their work.

The influence of physical effort in the production of an attack of angina pectoris, when the heart is ill-nourished by an imperfect blood-supply, is so well recognised as hardly to need mention. The relation of muscular effort to blood-pressure may undoubtedly become of pathological importance, especially in subjects who have passed the meridian of life, which period, however, is not to be stated in years. In advanced life nature demands and usually ensures that there be a tempering of the strain to the weakening powers.

It must, however, be admitted that in the majority of cases the effects of "strain" do not generally make themselves apparent unless there is also some other agent or agencies at work impairing the cardiac nutrition, or in some way rendering the tissues more vulnerable. In many instances careful investigation will show the patients to have been the subjects of anæmia, chronic alcoholism, toxæmic conditions, or nutritional derangements. In some instances an inherited cardio-vascular weakness may play an important part in precipitating the breakdown. The effects of fatigue during periods of convalescence, overwork at times of physical depression, and ill-regulated exercise in the presence of actual cardiac lesion, are only too frequently met with to permit of denial.

Strain is often said to be accountable for lesions of the aorta. Undoubtedly prolonged effort, such as is necessarily associated with certain laborious occupations, plays a very important part in the establishment of forms of chronic aortitis, particularly the variety usually described as "atheromatous," which, however, often implies interference with the coronary orifices and impaired nutrition of the heart muscle.

To sudden strain is also due the rare laceration of a degenerate myocardium, and possibly an acute dilatation of the left ventricle.

It is sometimes said that external violence is capable of producing rupture of a cardiac valve. Without absolutely denying such, the extreme rarity of the event warrants one in believing that it only occurs when the valve is already the seat of some morbid process, and even then it is extremely uncommon to meet with any such condition as could be considered due to external violence. Injury sufficiently severe to rupture a sound valve is almost invariably immediately fatal.

Latent disease is frequently made manifest by some form of undue or unaccustomed exercise. Not infrequently chronic cardiac pathological conditions are much aggravated by unwonted resort to procedures necessitating ill or unregulated effort. A hitherto efficient compensation may be completely ruptured by a trifling accident or a slight tussle. The importance of such considerations on therapeutical measures and medico-legal investigations will at once be evident.

It is not necessary to discuss here the particular localisation of the effects of strain. It will be sufficient to point out that those pernicious mechanical influences which, collectively, we may include under "strain" exert their power (i.) by establishing functional derangement (including leakage of valves and deficient emptying of chambers), which may be merely temporary or prolonged until organic defect is established; (ii.) by producing arterial valvular defects either as the result of dilatation of the aorta and its orifice, or through changes in the valves themselves.

(2) *Chemical.*—Agents conveyed in a soluble form by the blood to the various structures of the heart are accountable for many of the morbid conditions which form the pathological basis of cases of so-called cardiac failure, which, coming under the observation of the physician in their advanced and final stages present features more or less in common, and principally indicative of waning muscular powers. Certain of these chemical bodies are of autogenetic origin, as in Bright's disease, while others are heterogenetic, being manufactured outside the body, as for instance alcohol. The toxic agents resulting from microbial growth will best be referred to later.

Among the foremost of the toxic agents produced within the system must be mentioned those associated with the condition of *gout.* The influence of gout in producing functional derangement of the heart has long been recognised, but its most prejudicial effects are expended either directly or indirectly in establishing vascular changes, usually leading to increased cardiac pressure, and generally associated sooner or later with cardiac hypertrophy. Gout also seems to lead to degenerative changes in the coronary vessels, thereby producing an impaired nutrition of the muscular substance of the heart. The cardio-vascular associations of "gouty" kidney are not to be overlooked. It is very probable also that gout is capable of establishing primary degeneration in the muscle itself. Some of the cases of gouty syncope are probably due to such myocardial changes. It is sometimes said that gout is accountable for a form of chronic mural endocarditis, but such, if it ever occurs, is quite exceptional. The like establishment of an aortic valve disease (apart from mere aortic dilatation) is more probable.

Of even greater importance than gout stands out *rheumatism.* It is unnecessary here to consider whether the toxins associated with this disease are of microbial origin or dependent on morbid activity of the cells of the body; suffice it to say that rheumatism has a particular affinity for the serous membranes of the heart, and the majority of cases of diseased pericardium and endocardium are due to its influence. But rheumatism also very clearly exerts its influence on the muscular substance of the heart, and this seems to be particularly the case in young subjects. In not a few cases of acute rheumatism without endocarditis, or any exceptional degree of temperature, cardiac dilatation ensues apparently from the direct paralysing and degenerative influence on the protoplasm of the muscle cells.

In many of the so-called constitutional diseases cardiac affections arise, but in many instances it is impossible to distinguish between those dependent on impaired nutrition and those arising from toxic influence.

First amongst the pathogenic chemical agents of extrinsic origin must be placed *alcohol.* This body, especially in the form of beer, is accountable for a large number of cases of muscle failure occurring in adult labourers. The most characteristic cases of "alcoholic heart" are certainly met with in excessive consumers of malt liquors, or those who take their drinks "mixed," and where enormous quantities of drink are imbibed in the day while engaging in work of a heavy, often irregular and straining character. Some have thought that local differences in the manufacture of malt liquors may account for the seeming greater frequency with which alcoholic hearts are met with in some districts. Certainly in Manchester "alcoholic hearts" form a very large proportion of the cases of primary muscle failure, and occur particularly amongst market porters, barmen, brewers' draymen, cabmen, and labourers. "Alcoholic heart" is not commonly met with in women, who are less exposed to strain. It is interesting to note that athletes have long realised that beer was bad for their "wind."

Reference may here be made to the cardiac depressant action of certain animal and vegetable poisons, animal medicinal substances, such as thyroid preparations and numerous other well-known drugs. Suprarenal extract, however, leads to the development of high arterial tension. The deranging influence of tobacco is too widely experienced to need more than mentioning.

(3) *Thermal.*—Little need be said respecting the influence of heat and cold, light and darkness, on cardiac action and cardio-vascular structure. Exposure to extremes either of heat or cold produces a paralysing influence on muscle. Heat syncope and cardiac collapse from sunstroke are well-recognised clinical conditions. Pyrexia exerts a distinctly deleterious influence on cardiac muscle. The effects of cold in raising vascular tension must also be borne in mind.

(4) *Biological.*—Parasitic agents are very influential in establishing certain well-defined cardio-vascular lesions. The most striking example is

malignant, or, as it is now frequently termed, infectious endocarditis. Some pathologists look upon all rheumatic lesions of the endocardium as due to microbial infection. Inflammatory affections of the heart, particularly those of the pericardium and endocardium, occasionally occur in the course of scarlet fever, diphtheria, and sometimes in some of the other infectious diseases. Most if not all of the septic diseases seem capable of producing secondary affections of the heart.

The embryos of certain of the worms occasionally attack the heart. The cystic form of Tænia echinococcus may produce "hydatids" of considerable dimensions.

2. Predisposing Influences.—These may be considered according to whether they act through (*a*) the inherited or acquired textural characters of the individual, or (*b*) by modification of the environment.

(1) *Heredity.*—Certain families manifest a distinct tendency to early degenerative processes in their vessels, and to a premature cardiac regression. It is not necessary to discuss to what extent this is due to direct transmission. In some cases community of work and environment is a more reasonable and likely explanation. Family proneness to gout and rheumatism, and other conditions peculiarly liable to exert a baneful influence on the heart, must not be lost sight of. Cardiac failure from "vascular" causes oftentimes seems to "run" in families. As far as can be ascertained, race exerts comparatively little direct determining influence.

(2) *Sex.*—This is a factor of considerable importance, although the precise way in which the influence may be exerted is in many instances far from clear. The influence of occupation is closely allied with that of sex. Aortic disease is thus most frequently met with in men. Inefficiency of the aortic valves when occurring in females is almost always due to organic changes following rheumatic endocarditis or consequent on microbial infection. Mitral stenosis is much more frequently met with in women than men. Some have thought that this arises from a greater liability to rheumatism and chorea in the female. Primary muscle failure is generally met with in men. "Alcoholic heart" is rare in women. Dilatation from muscle failure and high arterial tension is, however, not unusual in women after the climacteric. Dilatation of the heart from chronic bronchitis and emphysema would seem to be equally common in the two sexes. The importance of chlorosis in young girls, by producing minor degrees of cardiac dilatation, must not be overlooked. In exophthalmic goitre or Graves' disease, most common in females, tachycardia and irregular cardiac action may be followed by evidences of dilatation and muscle failure. In modifying or increasing already existing physical cardiac deficiencies, the influence of pregnancy on the circulation, including increased arterial tension, must be remembered. Malignant endocarditis has been known to follow parturition.

(3) *Age or period of life* is a factor of great importance. Three periods may be recognised:—Firstly, that of *development* (including fœtal life), where maldevelopment and inflammatory processes along with their consequences may be expected. Secondly, the period of *maturity*, when the effects of strain and the influence of toxic agents, especially alcohol, are liable to be manifest, and when also the mechanical deficiencies of previous valvular involvement are apt to become apparent. Thirdly, the period of *decline*, when affections arising from malnutrition associated with vascular disease and degenerative changes are found.

Of diseases peculiarly prone to affect young subjects, and at the same time leave permanent crippling of the heart, rheumatism stands pre-eminent. Rheumatic fever and rheumatism in "silent" and easily overlooked forms, constituting little more than "growing pains," are very common in early life. This is also the period for the incidence of chorea, which is very often associated with endocarditis, and not infrequently with some degree of myocardial involvement.

Scarlet fever, and possibly some of the other infectious fevers, may exceptionally inflict injury on the endocardium in childhood. It would also seem as though a rheumatic endocarditis might be the first and only manifestation of the rheumatic poison. Speaking broadly, pericarditis and endocarditis are peculiarly the cardiac affections of the developmental period of life. The older a person grows the less liable is rheumatism, even though it occur, to inflict serious injury.

In adolescence, when activity and instability are striking features in the organism's development, so-called functional derangements readily arise.

In the period of adult life, as already indicated, the effects of strain on a healthy or already crippled heart, either from improper, unsuitable, or excessive work or ill-regulated exercise, commonly become manifest. In certain classes of the male community "alcoholic heart" is apt to be developed. The effects of other toxæmic states may become manifest, particularly those resulting from gout and Bright's disease. Syphilitic disease of the aorta and forms of atheromatous aortitis may by encroachment on the orifices of the coronary arteries or extension to the aortic cusps give rise to anginal attacks or the signs of aortic incompetence.

In the female, at both ends of the period of sexual activity, functional derangements are common. In chlorotic females a certain degree of cardiac dilatation is frequently detected. The influence of pregnancy, parturition, and lactation must not be lost sight of, especially in the case of a heart in any way crippled.

Mitral stenosis is the common lesion of adult females. Conditions of mitral regurgitation through a dilated auriculo-ventricular orifice, as the result of a rheumatic valvulitis, are not met with in either sex so commonly as was formerly believed, and certainly cases of mitral regurgitation through a dilated orifice with sclerosed segments are but rarely seen in the post-mortem room. The possible existence of mitral stenosis should always be carefully considered in every female presenting evidences of mitral incompetence and having an old rheumatic history.

In the advanced stages of middle life the tendency to general adiposity becomes apparent, and in many instances enormous deposition of fat in and around the heart impedes its action and impairs its nutrition, and not infrequently is associated with distinct degenerative changes which may lead to actual dilatation of the ventricular cavities.

In the later years of life processes of degeneration and infiltration lower the vitality and physical powers of both heart and vessels. Arterio-sclerosis paves the downward path. The coronary arteries are frequently the first to suffer, thereby diminishing or cutting off an adequate nutrition of the essential structure of the heart. Hence follow atrophy, fatty degeneration, fibrous transformations, and possibly aneurysm, softening, and it may be rupture of the ventricular walls.

In old people, although by no means limited to them, chronic Bright's disease often leads to associated cardiac hypertrophy, which sooner or later gives place to dilatation and its consequences.

In the years of diminishing vigour atheroma of the aorta is common, and either by extension of the atheromatous process to the aortic valves, or from enlargement of the aortic ring as a result, dilatation of the aorta and incompetence of the aortic valves may be produced. In cases of this kind the atheromatous condition may have extended to the coronary vessels, or at least have narrowed their orifices, thereby impairing the nutrition of the myocardium, and rendering adequate compensatory hypertrophy of the left ventricle imperfect or of short duration. At this time of life anginal attacks may occur.

(4) *Temperament.*—It is very difficult to decide to what extent "the sum of the physical peculiarities of an individual, exclusive of all definite tendencies to disease," should be allowed a place in the etiology of cardiac derangements. Temperament is not an easily estimated factor. Nevertheless in functional derangements of the heart it cannot be altogether laid aside, and even in organic disease has to be remembered when arranging a course of management.

(5) *Previous Disease.*—A former morbid process may (i.) by affection of the structure of the heart itself or (ii.) by producing impairment of tissues outside the heart have rendered the cardio-vascular apparatus more vulnerable or less able to meet the needs of the body and the requirements of daily life.

Some previous pathogenic influence may have led to injury, or established a process which, even when the particular etiological agent is withdrawn, leaves a textural scar or functional weakness which makes the affected tissues peculiarly liable to capitulate.

The most influential agent is rheumatism. This, especially when acting in the form of "rheumatic fever," commonly produces endocarditis, and frequently pericarditis. One attack is commonly followed by others, and whilst the heart is perhaps most liable to be damaged in the first, each subsequent exacerbation or fresh establishment of endocarditis renders the affected parts more predisposed not only to similar attacks, but to succumb to other pernicious influences. Thus in the case of "malignant" endocarditis it will often be found that the valves have been already crippled by a simple rheumatic process.

Scarlatina is said to precede pericarditis and endocarditis; but if all cases of rheumatism in scarlet fever be excluded, one is bound to admit that, considering the frequency of this infectious disease, it can only be accorded a very subordinate place amongst the affections predisposing to distinct cardiac disease.

Measles is also sometimes said to predispose to endocarditis, but this is doubtful.

Reference may here too be made to the influence of renal disease, especially when in the form of "granular kidney," in producing extensive cardio-vascular changes. In not a few cases the ventricular hypertrophy ultimately gives rise to a fatal dilatation.

(6) *Climate.*—Indirectly climatic conditions may be of considerable influence in the production and progress of cardiac affections.

The cardio-depressor action of cold is of much importance, and some are still found sufficiently loyal to ancient beliefs as to consider soil, temperature, and humidity important influences in promoting the incidence of rheumatic lesions of the heart. As might be expected, climate is chiefly of importance in cardiac etiology in so far as it is related to rheumatism. Rheumatic fever, however, would seem to be almost ubiquitous. The influence of malaria in the production of cardiac disease must also be noted.

(7) *Social position,* and all therein included, is to be credited with wide reacting influence for weal or woe in the production and development of cardiac disease. It will be sufficient to remind the reader that dilatation of the heart from laborious occupation or excessive beer-drinking, or the

two combined, is most frequently met with in the working class. Aortic regurgitation is common in men of all sections of society. It is generally thought that incompetence resulting from a dilatation of the aorta is commonest in working men, while that dependent on valvular sclerosis from the extension of an atheromatous aortitis is common in all ranks of life.

(8) *Education* must be allowed a place in considering the predisposing causes of cardiac disease. Not a few cardiac breakdowns date from a period of ill-advised and badly regulated physical education; especially is this of importance when athletic pursuits are strenuously continued beyond youth and early manhood. The importance of mental strain and emotional stress in the precipitation of functional derangements must not be overlooked.

(9) *Occupation.*—As above indicated, the "calling" in life is often of the greatest influence in determining the special form of cardiac disease.

Occupations of a laborious character will lead to hypertrophy, and if continued in states of malnutrition, with implication of the cardiac vessels, or after the limit of healthy compensation has been attained, dilatation and degenerative changes will inevitably result. The importance of work and pursuits necessitating sudden strain or intermittent stress cannot be overestimated.

Work requiring exposure to damp and cold and such influences as are supposed to lead to rheumatism indirectly become of some importance in the etiology of cardiac disease.

Duties or avocations making exceptional calls upon the nervous system are liable to produce functional derangements of the heart.

Sedentary workers, who feed not wisely but too well, frequently develop conditions associated with high arterial pressure, sclerotic changes in the coronary and other vessels, and degenerative changes in the myocardium. Ultimately such persons may suffer from cardiac dilatation, and present signs and symptoms practically identical with those so commonly seen in the over-worked and under-fed aged labourer.

(10) *Habits.*—From what has already been stated the bearing of habits on cardiac health cannot be forgotten. Bad habits, hygienically speaking, if they cannot be credited with directly engendering cardiac disease, at least oftentimes go far in exposing the subjects of them to the attacks of definite irritants.

Errors of eating and drinking must be held responsible not only for such conspicuous conditions as "alcoholic" and "gouty" heart, but for many manifestations of malnutrition and a considerable number of the so-called functional derangements of the heart.

Possibly the use of belts or constricting bodies may have exerted some influence, as was once thought, in the production of "soldier's heart."

Tobacco and indulgence in snuff and certain drugs also exert an indirect influence in deranging the cardio-vascular mechanism.

Reference may here also be made to habits which give rise to states of morbid excitement. In attacks of temper or conditions of worry or fright death may result from angina pectoris and syncope. Protracted emotion also exerts a prejudicial effect upon the heart and vessels. Sexual excesses may prove a not readily recognised cause of cardiac derangement.

II. MORBID PROCESSES

Before describing the individual lesions met with in the more common cardiac affections, it will be desirable to briefly consider the essential nature of the morbid processes affecting the cardiac structures. This constitutes what may be termed the general pathology of diseases of the heart.

(1) *Maldevelopment.*—The normal process of cardiac evolution may be arrested or deranged and malformation or malposition result. These can only be interpreted by a reference to embryology. Although it is manifest that most congenital cardiac deficiencies are dependent on some interference with the development of the fœtus, but little can be said respecting the actual etiological factors. The oft-quoted influence of maternal impressions is little more than mythical.

Congenital defects arising from fœtal inflammation are more easily understood. An inherited tendency to rheumatism or rheumatic fever in the mother may possibly lead to the establishment of endocarditis in the embryo, but here evidence is very uncertain.

A consideration of the individual forms of cardiac malformation does not come within the scope of the present article and has been discussed at page 455. It is, however, necessary to observe that at no period of existence can the cardiac textures be considered free from the attacks of pathogenic excitants, or incapable of reacting thereto. In studying the forms of cardiac disease originating after birth, it is necessary to remember that the predisposing influence of a developmental aberration may play a part. Thus malignant endocarditis not infrequently attacks a heart, the subject of some slight anomaly in the number or size of the valves, or in the character of the septum. A hypoplasia of the heart has been claimed as a common association of chlorosis, and explanatory of certain features of the anæmic heart, but it is well to remember that in anæmia the heart is very often dilated.

It only remains to add that congenital processes of disease, whether essentially of developmental origin or of the nature of a fœtal inflammation, are capable of initiating a series of conditions which usually declare themselves by such striking features as cyanosis, blood alterations, various nutritional derangements, abnormal cardiac action, and occasionally dropsy.

(2) *Anæmia.*—The integrity of the cardiac muscle is dependent upon an adequate supply of normally constituted blood. Conditions of cardiac bloodlessness may arise from local causes, or be a part of a general poverty of blood. In both cases the myocardium suffers. As it is the office of the blood to convey nutriment to the cardiac textures, states of anæmia if prolonged are apt to lead to degradation of structure and impairment of function.

In simple anæmia the heart may be markedly involved. As already indicated, the cardio-vascular apparatus in chlorotic girls is said often to be in a condition of subnormal development. The cardiac tissues are pale, often soft and flabby, diminished in size and consistency, and microscopically are usually found to be in a state of lowered activity, as indicated by the reaction of the protoplasm of the cells to staining reagents, or by changes indicative of its actual conversion into lower forms. In not a few cases of chlorosis distinct cardiac dilatation occurs.

In pernicious anæmia the regressive changes become conspicuous, and fatty degeneration of the cardiac muscle may be manifest to the naked eye, as the so-called "tabby-cat striation" or "thrush's breast" appearance. This irregular distribution contrasts strikingly with that of a localised anæmia due to limited vascular disease.

Anæmic conditions also tend to produce a condition of hydræmic dropsy, and occasionally not only may slight effusion be met with in the pericardial sac, but a somewhat sodden and œdematous condition of the cardiac textures generally.

Thus from general anæmia and its immediate consequences serious cardiac derangements ensue. Functional deficiencies are common, asthenic manifestations are easily brought out by slight effort, and dilatation of the ventricular cavities readily develops.

Anæmia of local origin is almost always dependent on impairment of the blood-supply from disease of the coronary arteries. At all events from a practical standpoint, the coronary arteries are to be considered as terminal vessels. This seems to be particularly the case as regards the important papillary muscles. Obstruction to the lumen, thickening of the walls, or compression of the vessels from without, inevitably lead to profound nutritional changes which, while starting as a simple local anæmia, quickly pass to more important states of degeneration or actual necrosis.

Anæmia of the myocardium is also said to occur from the pressure of pericardial effusions, and in conditions of acute and even chronic dilatation, but perhaps generally a state of local cyanosis is more apt to accrue.

(3) *Hyperæmia.*—Little can be said with certainty respecting the process of hyperæmia as it occurs in the cardiac structures, since post-mortem it usually leaves no distinctive characters, and clinically presents no special features. Some clinicians, however, believe that some of the temporary or so-called functional derangements of the heart may possibly be due to a state of local congestion. At the present time considerable doubt exists as to the occurrence of any condition meriting the term of general hyperæmia or plethora, although formerly many cardio-vascular derangements were so explained.

Passive hyperæmia of the cardiac walls may be said to result only as a part of a more or less general or secondary venous engorgement. Occasionally, however, in cases of acute dilatation, or rapidly established cardiac obstruction, as from the formation of a thrombus, it is not uncommon to find enormous distension of the venous sinuses, and extensive passive congestion of the heart and pericardium. The turgid myocardium has a dark, reddish brown appearance, and the veins, especially those visible in the subepicardial region, are tortuous, large, and engorged. The congested endocardium is slightly darker in colour, less glistening than usual, and the dark and hyperæmic muscle stretches the overlying endothelial lining.

(4) *Hæmorrhage.*—Small localised hæmorrhages into the cardiac tissues are not infrequently found post-mortem. In certain blood diseases, such as scurvy, purpura, infantile scurvy, profound anæmias, especially in the form termed "pernicious," and leucocythæmia, small punctiform hæmorrhages into the pericardium, or scattered through the myocardium, or even sometimes beneath the endocardium, are of common occurrence.

Associated with many of the infectious diseases, especially those of a septic nature, hæmorrhages into the cardiac textures are common. They are also not infrequently met with in certain cases of poisoning, particularly by arsenic and phosphorus.

In states where convulsions have occurred either from toxic influences, as in strychnine poisoning, or from local injury or disease of the nervous structures, small ecchymoses are occasionally found in the tissues of the heart at the autopsy. In conditions, also, where extreme venous engorgement has occurred, as, for instance, in the forms of suffocation, more or less extensive hæmorrhages, particularly into the pericardium, are common. In the final stages of many cardiac conditions small hæmorrhages frequently develop in the walls of the heart as well as in the body generally.

In some cases considerable areas of hæmorrhage may be met with. In an old man with advanced coronary disease we found a large area of hæmorrhage which had occurred into a myomalacic patch, and which, rupturing into the pericardium, led to a fatal issue. Hæmorrhagic infarcts of the myocardium result from lesions of the coronary vessels. Such vascular changes are usually of slow development. Occasionally emboli, particularly in cases of malignant endocarditis, cause acute blocking, with rapid effusion of blood into the adjacent muscles. Aneurysm of the coronary artery may result. The rupture of a cardiac aneurysm is one of the well-recognised causes of hæmo-pericardium and sudden death.

Hæmorrhages into the cardiac structures are usually situated chiefly in the pericardium or endocardium, or in the tissues immediately beneath. They are generally of very limited extent, and should the patient recover from the condition giving rise to the hæmorrhage, the effused blood is doubtless readily absorbed. Ecchymoses of the pericardium and endocardium must not be confused with post-mortem staining.

Occasionally hæmorrhages into or beneath the endocardium occur in the region of the valves, particularly in connection with infective or malignant endocarditis.

Extensive areas of hæmorrhage into the cardiac substance may be met with in fatal cases of violence. Sometimes, especially in children, much bruising of the heart may occur without rupture into peri- or endocardial cavities.

(5) *Dropsy.*—An œdematous condition of the cardiac textures is always a secondary process. Such occasionally occurs in Bright's disease, and to a less extent in certain hydræmic states of the blood. Not only may there be transudation into the pericardial cavity, but the sub-pericardial tissue, and even the myocardium, often assume a boggy and water-logged appearance. Frequently the surrounding adipose tissue will be found distinctly œdematous. Where large pericardial adhesions exist, the old inflammatory tissue not infrequently presents considerable œdema. Also in the final stages of cardiac disease the heart's tissues participate with the rest of the body in a more or less extensive œdema. This is particularly marked in cases of primary muscle failure. The myocardium, however, even in these cases, from the character of its structure and the nature and arrangement of its fibres, presents but little evidence of extensive serous transudation.

(6) *Thrombosis, Embolism, and Infarction.*—Local clotting may occur either in the cavities of the heart or in the vessels supplying the heart.

The cardiac thrombus may be of acute or chronic formation. It is frequently met with in cases of dilated heart, occurring more particularly in the appendices of the auricles, which are commonly found completely obliterated at the autopsy by firm and often laminated clot. Thrombosis also occurs between the trabeculæ, and sometimes even at the rounded apex of a dilated ventricle.

Thrombosis also constitutes an important process in the formation of extensive vegetations, as occur in such abundance and in so exuberant a form in malignant endocarditis.

By slow deposition of fibrin a thrombus firmly fixed to the endocardial wall may extend forward into the auricular or ventricular cavity, and thus give rise to a "cardiac polypus." When formed in the auricle it may project sufficiently to obstruct the auriculo-ventricular orifice, and give rise to symptoms of valvular disease. They have even been seen as ball-like masses detached from the cardiac surface.

Thrombosis of the coronary arteries is an extremely important condition, cutting off, as it necessarily does, the nutritional supply to the cardiac muscle. It is usually dependent on changes in the vessel walls, arising as a part of a general arterial sclerosis, or sometimes from local atheroma, arteritis obliterans, or simple senile calcification. Occasionally the thrombus is secondary to embolic obstruction, due to detachment of vegetations from the aortic valve or aorta in malignant endocarditis or aortitis.

The endocardium is the home of emboli. It is in the cavities of the heart or on the surfaces of the valves that most of the emboli arise, which presently, we shall have to show, form such important factors in the secondary lesions of cardiac disease.

Cardiac infarction results from obstruction of a coronary artery. Should the occlusion or obliteration of the vessel be associated with extravasation of blood, a so-called "red" or "hæmorrhagic infarct" results. It is important to remember that while embolic blocking of the coronary vessels is comparatively rare, obstruction from sclerosis, atheroma, calcification, and thrombosis is common. In the latter conditions the myocardial areas, rendered anæmic, and becoming the seat of simple softening, constitute a "white" infarct or area of cardiomalacia. The red infarct will present a dark-red appearance or mottled aspect, varying from brown to yellow. Sometimes the central parts are white or yellowish white, and the borders red or brown. After a certain amount of absorption or disintegration of the blood has occurred, the infarcted tissues often assume a yellowish-gray appearance, or in old cases a grayish, translucent, structureless aspect. In some instances the myocardium may present a depressed puckered scar at the seat of the old infarct.

Sometimes the softening, resulting from coronary obstruction, may involve one or more of the papillary muscles, rendering them ineffectual as contractile structures. The softening may also involve the overlying endocardium, and extensive clotting will then occur over the affected areas. The whole depth of the myocardium may even be involved, and rupture occasioned into the pericardial cavity.

The importance of the above processes in the production of fibroid induration of the heart will be referred to later.

When the embolus contains pyogenic organisms, septic myocarditis or an acute abscess of the cardiac walls may result. This rupturing into the pericardium may produce acute septic pericarditis, or, extending into the cavities of the heart, give rise to a general pyæmia.

(7) *Inflammation.*—It is not our purpose here to discuss the nature of the inflammatory process, or the exact relationship of the lesions found to the etiological factors present. Suffice it for the present to indicate the main outlines of the leading characters of such changes as custom and experiment have recognised as inflammatory. The essential features of the inflammatory process may be divided into those of an exudative and those of a proliferative character. These differences would appear to depend, first, upon the nature and intensity of the irritant; and, secondly, on the structure, especially as regards vascularity, of the tissue involved.

The inflammatory process as it occurs in the heart may be limited to either serous, muscular, or connective tissues, although frequently involving all structures. In studying the phenomena of cardiac inflammation due consideration must be given to the anatomical and physiological peculiarities of the affected parts. The exposure of the endocardium to ready infection from irritants in the blood stream; the non-vascular character of the inner lining of the heart and the greater part of the valves; the dependence of the myocardium on the integrity of the blood-supply; the liability of the extension of inflammatory processes to the pericardium and other cardiac tissues; and the influence of the movements of the heart, constitute factors having important bearings on the inflammatory process.

In acute inflammation the exudative factor is most marked. The pouring out of coagulable serum, the passage of leucocytes, and the escape of a variable number of red corpuscles, are the chief of the vascular phenomena. At the same time textural changes occur, chiefly of a degenerative character; but in the less acute forms indicating efforts at a reactive process, in which the connective tissue cells play an important part. Should the inflammatory process affect pericardium or endocardium the products are enabled to escape from a free surface. In the former they collect in the pericardial sac, but in the latter they may be conveyed by the circulation to all parts of the body, thus readily explaining the general toxæmic manifestations met with in certain forms of endocarditis. In myocarditis the irritant must reach the muscle through the coronary vessels—at present we know but little as to the lymphatics of the heart in pathological states—or else, as often seems the case, by extension from the peri- or endocardium. Here the products of the inflammatory process cannot escape, and either by direct pressure, or the influence of morbid products on the several elements, lead to various degrees of degeneration or actual necrosis. In the less acute and chronic forms of inflammation the proliferative changes become conspicuous. Multiplication of the connective tissue and endothelial cells leads to the production of tissue which, however, is to be considered as of a decadent type. In the endocardium, and especially in the valves, increased production of tissue, usually of a fibrous character, leads to extensive mechanical defects, as will be indicated in the section on valvular diseases. In the pericardium local or general thickening may result, and frequently the pericardial sac is obliterated by the formation of adhesions. In the myocardium the intermuscular tissue may be increased in amount and rendered of a more fibrous character, and compress or even actually replace the essential muscle elements. Frequently evidences of degeneration and destruction occur in the same case with proliferative changes.

The so-called *infective granulomata* may conveniently be referred to here, closely allied as they are to the process of inflammation as we are accustomed to view it. The specific irritants of tuberculosis, syphilis, and actinomycosis occasionally invade the cardiac tissues. Tubercular lesions are by no means uncommon in the pericardium. Sometimes large cheesy nodules occur in the subpericardial structures, or even in the myocardium. At times more or less diffuse caseous areas are met with, or a certain degree of generalised fibrosis. Gummata are rare. Exceptionally they develop as caseous-looking yellowish-white nodules, often surrounded by dense fibrous tissue. They may even rupture into the cavities of the heart. Multiple gummata have been found in hereditary syphilis. Syphilis may also give rise to a diffuse interstitial formation of fibrous tissue, and some have claimed that this should be considered an arterio-sclerotic induration rather than a true interstitial myocarditis.

Actinomycosis is characterised by the formation of gray or yellowish-white granulomata which may suppurate, and in which the typical organisms may be found.

(8) *Atrophy.*—This constitutes the simplest form of regressive interstitial change. It may affect the heart as a whole, and be a part of a general atrophy of the body, such as occurs in starvation and diseases like phthisis and diabetes; or it may be local, or at least affect the heart to a greater extent than the rest of the body. Usually the most conspicuous change is in the muscle cells.

Simple atrophy may arise from practically any condition which deranges the normal equilibrium between the evolution and involution of the individual cell. It is of common occurrence in states of premature decay, in all wasting affections, particularly malignant disease and pulmonary tuberculosis, and in affections generally speaking associated with impaired nutrition.

The heart has a small shrunken appearance. The muscle cells are diminished in size, and usually darker in colour from the presence of yellow pigment granules. In advanced forms met with, particularly in senile and cachectic conditions, it may assume a distinctly brown aspect, when the condition is commonly termed "brown atrophy" or "pigmentary atrophy." Non-ferruginous granules become deposited in the muscle cells tending to congregate at the extremities of the nucleus.

(9) *Hypertrophy.*—Cardiac hypertrophy must be viewed as rather a physiological than pathological process. True hypertrophy of the heart's muscle is always a compensatory measure. It is nature's method of assuring adaptation. It is the reaction to influences making for a disestablishment of cardiac equilibration.

Hypertrophy of the heart frequently arises from the influence of persistent effort, such as is necessarily associated with many forms of laborious work. Such reaction to a physiological over-activity is well seen in blacksmiths and professional runners or rowers. A slight amount of hypertrophy is said to occur normally in pregnancy, possibly as a consequence of increased arterial tension.

Increased activity of the heart from nervous causes may lead to a certain degree of increased muscular development, as is seen in cases of Graves' disease, and so-called functional tachycardia. In most of these instances, however, dilatation seems to precede hypertrophy.

The most marked degrees of hypertrophy are found in conditions of valvular deficiency and states of high arterial tension.

The causes of cardiac hypertrophy may thus be conveniently divided into (i.) intrinsic and (ii.) extrinsic, according as to whether they arise in the heart itself or originate without the heart.

As will be shown when considering the lesions of the different valves, both insufficiency and stenosis may lead to conspicuous hypertrophy. Generally speaking, in the latter form of lesion, since it is usually of slow development and of only gradually increasing intensity, and because also the effects of the mechanical defect become chiefly manifest on the walls of the cavity immediately behind the obstruction during systole, hypertrophy will be the first and earliest secondary change, and probably the most persistent. While in valvular incompetence, especially when rapidly established, dilatation is usually the first and most noticeable effect, while hypertrophy is only a later and often less marked effort at readjustment. Impediment to the free action of the heart may lead to considerable hypertrophy, as is frequently seen in "adherent pericardium." Probably here dilatation precedes hypertrophy. Frequently also there are associated endocardial lesions. In conditions of chronic renal disease, particularly in the form of "granular" kidney, very extensive hypertrophy occurs. The increase in muscular development affects chiefly the ventricles, and mainly that of the left.

It may be interesting here to observe that in old age the heart is often found increased in size. Possibly this may be best explained by a reference to the condition of the vessels.

Aneurysm of the aorta is not necessarily associated with any marked degree of hypertrophy. When, however, it leads to any appreciable degree of obstruction which is quite exceptional, or when by involvement of the first part of the arch incompetence of the aortic valve is occasioned, enormous increase in the extent of the walls of the left ventricle may be found.

In pulmonary obstruction, such as in cases of chronic bronchitis, emphysema, fibroid conditions of the lungs, marked hypertrophy of the right ventricle occurs, which, however, is not limited to it, but, as we should expect from a recognition of the solidarity of the heart, the left-sided cavities also participate, although to a less extent.

"Idiopathic" hypertrophy is practically an unknown and rightly discarded condition.

Hypertrophy of the heart has been divided into (i.) simple, (ii.) eccentric, and (iii.) concentric. It is doubtful if the last form ever occurs. In clinical and pathological practice the second form, or hypertrophy with dilatation, is usually met with.

The so to speak mere mechanical necessities play a determining part. Thus in aortic stenosis almost pure hypertrophy often occurs. In aortic incompetence, since the left ventricle fills from two sources at the same time a double influence must be considered—one destructive, the other constructive. Hypertrophy, however, can only be maintained when there is adequate nutrition and the expenditure kept well within the cardiac income. This overstepped dilatation becomes the source of cardiac downfall.

It must also be remembered that when hypertrophy is associated with marked dilatation, the larger the cavity the greater the contractile power required to empty it.

True hypertrophy consists in an actual hyperplasia of the individual muscle cells. In many cases, and especially in young subjects, a numerical increase also occurs.

All parts of the heart's muscle share in the hypertrophy, though seldom equally so. The ventricles show the most conspicuous increase in size. The papillary muscles are often greatly hypertrophied. All degrees of hypertrophy are met with. Occasionally the heart doubles both in size and weight.

(10) *Degenerations.*—Impaired nutrition of the cardiac tissues leads to degenerative processes. The normal protoplasm suffers regressive change, and is converted into less highly developed forms. Degeneration of the cardiac structures is always to be looked upon as an involution and strictly pathological. Hard and sharp lines, however, cannot always be drawn between the different varieties, which, in not a few cases, tend to pass from one to the other. Still, generally speaking, and for purposes of description, certain more or less distinct forms may be recognised.

Parenchymatous Degeneration or Cloudy Swelling.—This forms the simplest of regressive changes. It is characterised by a swelling up of the cells and fibres, the protoplasm becoming cloudy, and forming fine free granules with indistinctness or disappearance of the nuclei.

The micro-chemical reactions of the granular material show it to be albuminoid. Should the process be arrested at an early stage the affected cells apparently recover. This possibly explains the perfect restoration which occurs in not a few cases of slight degrees of dilatation of the heart.

This form of degeneration is met with particularly in the cardiac muscle cells, and occurs as a result of toxic conditions. It is common in most of the infectious diseases. In typhoid fever, diphtheria, and septic processes the morbid change is usually well marked. It is also met with in acute Bright's disease.

Fatty Degeneration.—This is one of the most frequent pathological conditions affecting the myocardium. It is essentially a different process from fatty infiltration, although the two often occur in combination. It is characterised by a conversion of the cellular contents into fat elements. The change occurs at the expense of the cell protoplasm. It may take place throughout the myocardium, but is more frequently patchy in distribution. The cells have an indistinct granular appearance due to the accumulation of minute fatty globules. In advanced cases the nucleus also disintegrates. Osmic acid stains the fatty granules black, and the cell assumes a vacuolated appearance when the fat is dissolved out by ether.

The macroscopic characters of the tissue differ somewhat according to the extent and duration of the change. The " tabby-cat striation " so common in pernicious anæmia is due to a patchy distribution which occurs apparently quite irrespective of any alteration in the local vascular supply.

Fatty degeneration occurs in a large number of conditions; in fact, any prolonged morbid state leading to general impairment of nutrition is liable to produce it. It is common in all forms of anæmia, but particularly marked in the "pernicious" variety, and in toxic states, such as from phosphorus, arsenic, or alcohol. It occurs more or less naturally as part of the general disintegration of old age, but here probably at least for the most part in association with a certain degree of actual vascular disease. In pyrexial and toxæmic conditions, especially those associated with the infectious fevers, it is common. Some cases of fatal cardiac syncope in diphtheria are due to this cause. It also occurs in various wasting diseases, and is frequent in cancer, chronic tuberculosis, and all forms of cachexia and inanition. Fatty degeneration may also result from local disturbance of the cardiac nutrition as from long-continued increase of arterial pressure, and especially when this leads to passive venous congestion. The anæmia and atrophy, secondary to coronary obstruction, usually also proceed to fatty conversion of the muscle cells in the affected area.

Hyaline degeneration seems to be dependent on some process akin to coagulation, whereby the cell contents assume a vitreous, shining, translucent appearance. The product formed is a strongly refracting substance and very resistant body, being insoluble in water, staining yellow with iodine, but giving no reaction with chloroform or ether. The fibrous tissue of the vessels are specially affected, but the muscle elements may also manifest the change. The affected textures macroscopically have a distinctly cloudy appearance. As to its exact pathology there is much dispute, but except that it is undoubtedly a manifestation of a regressive metabolism of the cell little can with certainty be said. It occurs particularly in toxæmic conditions.

Hyaline and granular degeneration frequently occur in association with other regressive nutritional processes.

Dissociation of the muscle cells, which has been described as "segmentary myocarditis," occurs in several conditions of depraved nutrition, and is sometimes met with in cases in which hyaline and other forms of degeneration occur. Some have contended that this loosened and separated condition of muscle cells is dependent on a post-mortem change.

Mucoid degeneration is occasionally met with in the connective tissues of the heart, occurring perhaps most frequently in the thickened prominences of sclerosed valves. The affected patches have a gelatinous appearance.

(11) *Infiltrations.*—Fundamentally the process of infiltration is quite distinct from that of degeneration, although frequently found in association. Both are to be considered as evidences of impaired nutrition.

Calcareous Infiltration.—Deposits of lime salts are of very common occurrence in connection with the cardio-vascular mechanism. Usually they are met with in the form of carbonates and phosphates, but sometimes in combination with salts of magnesium. All parts of the heart may be involved. Occasionally calcareous plates are found in old pericardial adhesions.

Sclerosed valves commonly present extensive deposition. The coronary vessels are frequently involved, and in some few instances even the myocardium becomes the seat of a plentiful deposition of lime salts.

Calcareous change is particularly liable to affect the valves, and is an important factor in producing and maintaining forms of valvular obstruction. In sclerosed valves large, hard nodular or irregular masses are very common, and go far to render the segments immobile.

Calcareous deposition is said to occur primarily as a manifestation of old age, but it will usually be found to have taken place in structures already the seat of degenerative processes. The true pathogeny of the condition is by no means clear. Apparently local lesions present conditions which predispose to or actually produce a precipitation of the lime salts or a conversion of the soluble into insoluble compounds.

Uratic deposition is said to occur within the cardiac structures in gout, but if such is ever the case it is quite exceptional.

Lardaceous infiltration also affects the heart as a part of a more general involvement. It cannot be considered as of any special clinical importance. The coats of the coronary vessels are earliest affected, but in advanced cases the basement membranes, fibrous elements, and even muscle cells may be affected. As is well known, it occurs particularly in association with chronic syphilis and states accompanied by long-continued suppuration.

Fatty infiltration frequently occurs in combination with fatty degeneration. As already indicated, they are, however, essentially different processes, the former characterised by a mere cellular deposition, the latter arising from actual protoplasmic conversion.

Fatty infiltration or cardiac lipomatosis is commonly but a part of a general obesity. Deposition of fat occurs particularly in the pericardium, the subepicardial tissue, in and along the septum, and around the base of the heart. The whole organ may be enclosed in a panniculus adiposus. Where excessive the cardiac action may be much impeded, and atrophy and degeneration of the muscle cells not infrequently result. Rupture of a fatty heart has occurred spontaneously from a comparatively trifling cause.

This condition of fatty infiltration arises either from increase of intake or diminution in expenditure. It thus occurs in large eaters, chronic alcoholics, or in states where, as in some forms of anæmia, there is diminished oxidation.

Certain families, and women particularly in advanced middle life, are peculiarly prone to develop this condition.

(12) *Necrosis* forms the final and most serious form of the degradation processes. Actual death of cardiac tissue may occur in several forms:—

Simple softening, constituting cardiomalacia, occurs as the result of obstruction of a coronary vessel from a mechanical or non-septic cause. When a large branch is involved, and consequently a wide area of muscle rendered anæmic and subsequently necrotic, death may occur from rapidly established cardiac failure. In other cases the softened area gives way, and allows of rupture of the cardiac wall, with escape of blood into the pericardium. When the softened area is absorbed and replaced by scar tissue, a cardiac aneurysm may result.

Ulceration or molecular necrosis constitutes a very important feature of malignant endocarditis. Here the microbial invasion, or the toxins resulting from their growth, produce extensive death of the structures, forming the affected valve or mural endocardium. The necrotic tissue becomes entangled with blood and fibrin, increasing the production of the abundant vegetations so characteristic of this form of endocarditis; softens into minute granules, which are washed away in the blood-stream; or, being detached in fragments of varying size, are carried along and lodged in some distant vessel as emboli. Such accidents, however, are not limited to ulcerative endocarditis, as will be shown presently. Sometimes in septic endocarditis the valvular tissue yields, so producing an acute aneurysm of the valve.

Occasionally necrotic foci are met with in the myocardium due to the penetration of organisms from the endocardium or their conveyance by means of the coronary vessels. An embolic plug, proceeding from vegetations of a malignant type, may lead by a process of rapid softening to dilatation of a coronary artery and the formation of an acute aneurysm.

Gummatous and caseous necrosis are only very exceptionally met with in the substance of the heart.

(13) *Tumours.*—Primary growths of the heart are very rare, and little more than pathological curiosities. Sarcoma, fibroma, myxoma, and myoma have all been described. Some are said to be of congenital origin. Occasionally they assume a polypoid form, and extending into the cavities of the heart may produce cyanosis, and give rise to evidences of cardiac obstruction and muscular deficiency.

Secondary growths are more frequent. They may either be of a carcinomatous or sarcomatous type. Melanotic sarcomata occur sometimes as extensive and plentiful secondary depositions in the myocardium. Secondary nodules of growth may be found embedded in the walls of the heart or projecting from its inner and outer surfaces. Sometimes sarcomata extend directly to the pericardium and heart from the mediastinum. Cancer of the œsophagus very exceptionally extends to the heart.

(14) *Parasites* of the heart are very rare in this country. The cysticercus form of the *Tænia solium* is occasionally met with, and is said to occur most frequently in the walls of the left ventricle. It varies in size from a pea to a marble. Hydatids, or the cystic form of *Tænia echinococcus*, may develop into cysts of considerable size. When rupture occurs into the cardiac cavities embolism of the pulmonary or systemic vessels may result.

These parasitic involvements of the heart are of but little clinical importance, and do not call for further consideration here.

III. Effects of Cardiac Disease

Many of the secondary results of cardiac disease are chiefly of importance from their clinical significance, and are best dealt with from that standpoint. For the present purpose it will be sufficient to indicate only the leading pathological features of practical interest.

Passive venous congestion is one of the most important of the consequences of cardiac failure. Its essential character is blood stagnation.

Cyanosis.—Every venously congested tissue tends to assume a deep red, livid, or even bluish tint. The condition arises from any cause impeding due aeration of the blood. In many forms of congenital defect of the heart it is a specially conspicuous feature (morbus cæruleus). In more or less all cases of cardiac failure, when the right cavities become distended cyanosis appears, and deepens in intensity with the progressing embarrassment of the pulmonary circulation. Cyanosis is especially marked in many cases of mitral stenosis. It is also a conspicuous feature in most cases of primary muscle failure.

The cyanosed tissues vary in colour from a slight dusky red to a deep purple-black. The most intense degrees are best seen in peripheral parts of the circulation.

Passive Hyperæmia of the Lungs.—Passive pulmonary congestion is sometimes associated with pulmonary œdema. Venous engorgement may, however, be an early result of cardiac disease and long maintained as almost the only serious effect. It is met with in its most marked form in mitral stenosis, in which the pulmonary tissues may be subjected to passive hyperæmia for years before the failure of the right ventricle allows establishment of a general venous engorgement. This increase of pulmonary pressure is often in cases of mitral disease relieved from time to time by hæmoptysis, which may thus prove a beneficial accident in the young subjects of mitral stenosis.

As the result of long-continued passive congestion the lungs may pass into conditions of so-called "red" and "brown induration." A cyanotic lung is of increased consistency, its elasticity diminished, and generally increased in weight, though often lessened in size. In colour it varies from a red to a deep brown, according to the degree and duration of the engorgement. The connective tissue is usually increased and the air vesicles diminished in size, while their walls are thickened. The capillaries are much distended, tortuous, and hæmorrhages readily occur.

"*Nutmeg*" *Liver.*—The hepatic tissue is one of the first to fall under the influences making for passive hyperæmia. Failure of the heart to empty its right-sided cavities necessarily interferes with the return of the blood from the inferior vena cava. Passive hyperæmia of the hepatic veins ensues, and if prolonged leads to such changes in the appearance of the liver substance as is aptly characterised by the terms "myristicated" or "nutmeg." The organ is often enormously enlarged and much increased in weight. In states of venous congestion the sponge-like adaptability of the organ becomes of considerable practical importance. The liver substance is dark-red in colour, and, as seen through the smooth and stretched capsule, has a mottled appearance. On section the large veins are found distended, but after death the alteration in blood-pressure allows the thin-walled vessels to collapse and the liver to shrink to a considerable extent, so that frequently the pathologist finds an organ which does not correspond in size to that mapped out during life.

The cut section of a "nutmeg" liver presents a mottled appearance; the central part of each lobule is of a dark reddish-brown colour, due to distension of the intra-lobular vein and engorgement of the capillaries, with atrophy of the adjacent liver cells. The peripheral zone of the lobule is o a gray or yellow colour, due to extensive fatty changes in the liver cells and in advanced cases the atrophy, degeneration, and pigmentation wit bile may be extensive.

"*Cyanotic*" *Spleen.*—The spleen participates in the results of obstructio to the portal blood-flow, hence, in most cases where the effects of impede blood-flow from cardiac disease are transmitted to the liver, the splee sooner or later participates in the portal venous engorgement. It i interesting to note, however, that the capillaries of the liver may long suc cessfully act the part of a protective buffer to the portal system. A cyanot spleen is of a dark purplish-red colour, firm in consistency, tough, and n readily friable. It is generally enlarged to some extent, but not always s In cases of congestion from hepatic cirrhosis the spleen is usually muc larger and heavier than when of "cardiac" origin.

Passive congestion of the gastro-intestinal tract is usually well mark in cases dying from progressive heart failure. The veins of the stomach and intestines are engorged, the mucous membrane swollen, and frequently small hæmorrhages are present. It is important to note that cyanotic conditions of these structures readily pass into a state of actual inflammation. It is quite common to meet with evidences of gastro-intestinal catarrh in fatal cases. Hæmorrhoids and sometimes œsophageal varices may occur.

Passive venous congestion of the pancreatic and other branches of the portal vein lead to impairment of the function of the organ concerned, and the engorgement of the peritoneum leads to the establishment of ascites; and in long-standing cases chronic indurative processes seriously damage the absorptive properties of the peritoneum. Special note should be made of the porcelain-like thickening of the capsules of the liver and spleen, and other parts of the serous membrane of the abdomen met with in long-standing cases of ascites of cardiac origin.

Cyanotic kidneys are met with in all cases of long-continued heart disease. The organs are enlarged, increased in weight, of firm consistency, having a dark brownish-red colour and with prominent stellate veins. The capsule may not separate very readily, but the surface is generally moderately smooth. Occasionally old scar-like depressions are met with. Both cortex and medulla are increased in extent, of a dull red or purplish colour, with the venæ rectæ and glomeruli often prominent. Microscopic examination shows great distension of the veins and usually distinct changes, mainly degenerative, in the renal epithelium. In many instances it is clear that no hard and sharp line can be made, either pathologically or clinically, between states of venous stasis and actual inflammation. It is certain that in the kidney conditions of the former readily merge into those of the latter. In cyanotic kidneys minute hæmorrhages are frequently found, as also pigment granules in the cells, and not uncommonly hyaline casts in the tubules.

Congestion of the genital organs, when occurring in the female, may be of importance in explaining the occurrence of menorrhagia or metrorrhagia.

Passive hyperæmia of the superior vena cava and its branches is manifested by engorgement, and sometimes by tortuosity of the veins of the neck and face. Special reference may here be made to certain curious cases of "alcoholic" heart, which clinically much resemble cases of mediastinal tumour at the first glance—the venous engorgement and dropsy of the head and neck and upper part of the trunk and upper extremities being often extreme.

The brain and its membranes are also involved in the venous distension, and this explains many of the troublesome cerebral manifestations met with in cardiac cases.

Local anæmia occasionally occurs as a seeming consequence, or, at least, accompaniment of cardiac disease. In malignant endocarditis general anæmia of rapid production may be very marked. Mitral stenosis is sometimes found in young chlorotic females. Cardiac dilatation is by no means exceptional in simple anæmia. A fatty and anæmic heart is sometimes the only explanation of a fatal syncope.

Dropsy forms one of the most important consequences of cardiac failure. Serous transudation may occur into the subcutaneous tissues constituting anasarca, into the texture of organs as in pulmonary œdema, or into serous cavities forming hydrothorax, hydroperitoneum, and ascites. Several factors play a part in its production. Mechanical obstruction to the return of venous blood, alteration in the walls of the blood-channels, depraved conditions of the blood and derangement of the lymphatic circulation, may all be of importance. The first, however, forms the most evident explanation. Impediment to the return of venous blood is, as already indicated, the most constant and striking feature in cardiac failure. Generally speaking, when dropsy occurs it is more or less proportional to the degree of venous engorgement. This, however, is not always so. In many cases very advanced degrees of cardiac disease may be attained without any sign of dropsy, although it may appear almost suddenly shortly before the end. Hence, although there is some evidence in favour of the contention that no direct relationship exists between the amount of heart disease and dropsy, it must be insisted upon that usually dropsy develops with the failure of the vigour of the cardiac muscle, and in most instances is essentially proportional to and progresses with the muscular embarrassment.

It will be shown elsewhere how readily a heart in unstable equilibrium may be influenced by comparatively trifling agencies, and the adjustments of compensation which depend on muscular adaptiveness may be suddenly upset, so allowing of the rapid development of dropsy. A readjustment may be followed by as rapid a disappearance of the transuded fluid. The importance of such predisposing influences as a severe accident, mental shock, acute disease, and the like, cannot be over-estimated in clinical work. Mechanical conditions, which in health would have no appreciable influence, may in cardiac derangement prove sufficient to determine the occurrence and regulate the distribution of serous transudation. The common occurrence of œdema of the feet and legs in early or slight forms of cardiac asthenia are sufficient proof of this.

But a merely mechanical view is far too limited to explain all the causation of dropsy in cardiac disease. Disturbance of the nervous mechanism, altered nutrition of the walls of vessels, impoverished conditions of blood, will lead to transudation, and in some heart affections there is reason to believe that some distinct derangement of a peripheral mechanism may be the true explanation. It is only by admitting such a view that one can explain the curious localisation of œdema—as, for example, that over the sternum and sacrum, or even into the scalp, such as is by no means exceptional in cases of alcoholic muscle failure.

Comparatively little, however, is known as to the local conditions making for an increased permeability in cardiac conditions. In long-continued states of vascular derangement nutritional changes must of necessity occur in the vessel walls, and probably tend greatly to maintain tendencies to transudation.

It must also be remembered that in most cases of cardiac failure there are influences greatly impairing the normal lymphatic circulation, since the integrity of the latter is necessarily in great part dependent on the perfect adaptability of arterial and venous blood-pressure.

It must also be admitted that anæmia may be a factor in the subjects of ordinary valvular heart disease, so to speak, promoting œdema.

The dropsical effusion is usually a transparent yellow or yellowish gree fluid, with few or no cellular elements, showing no tendency to spontaneou coagulation, of low specific gravity and containing but little albumin or othe solids. The fluid, however, probably varies in character, as would seem t be sufficiently shown by the free running or otherwise through a Southey' trochar.

Œdema of the lungs is of special importance in heart disease. Usually develops in organs the nutrition of which has been long impaired. Occ sionally it arises with startling rapidity. The lungs are large and bulk much increased in size and weight, pale, and pit on pressure. On section clear, frothy, serous fluid escapes often in enormous quantities.

Thrombosis, Embolism, and Infarction.—Thrombosis may occur as a consequence of endocarditis and morbid changes in the lining of the heart and vessels. It is common in dilatation of the cardiac cavities, and also arises secondary to embolic obstruction. Reference has already been made to the occurrence of thrombosis in the cardiac cavities and coronary vessels Local clotting also sometimes occurs in heart cases in the peripheral vessels when the propulsive force of the organ has suffered much diminution. Occasionally local œdema of an extremity, developing as a terminal condition, is found to have been due to a thrombus. Local coagulation may affect either the arteries or the veins; when of embolic origin it is of course always arterial. The medium-sized vessels seem to be most prone to be involved.

The embolus occurring in cardiac disease is commonly formed by blood-clot, but sometimes it may arise from a detached fragment of a calcified valve, a portion of vegetations, or parts of a softening thrombus. In endocarditis embolism is common. It also frequently occurs in cases of dilated heart, and particularly in such cases of valvular disease as mitral stenosis, where cardiac thrombi are especially liable to form between the musculi pectinati of the left auricle, in the left auricular appendix, and around and between the columnæ carneæ of the left ventricle. The manifestations of embolism vary according as to whether the obstructed vessel is furnished with a free anastomosis or is terminal. In the former the effects are usually temporary and comparatively trivial. Obstruction of so-called end arteries, such as the pulmonary, renal, splenic, and coronary arteries, the central artery of the retina and the nutrient arteries of the brain, to which may also be added for practical purposes the superior mesenteric artery, lead to results more or less serious, and frequently producing permanent defect. The consequences of such blocking are engorgement, hæmorrhage, or necrosis occurring separately or in combination.

Acute aneurysms may arise in the cerebral and coronary vessels as a result of embolic blocking in malignant endocarditis.

Hæmorrhagic infarction of the lungs is an almost constant accident of severe cardiac disease. The affected portion, varying in size from a minute patch to a mass involving the greater part of a lobe, is rendered solid, of a deep red colour, and often wedge-shaped. The lung alveoli are distended with blood, and the adjacent vessels engorged. Frequently an "embolic" pneumonia in the neighbourhood of the infarct greatly extends the area of consolidation. Some pathologists claim that many of the pulmonary infarcts are not of embolic origin, but are to be looked upon as true hæmorrhages.

Renal infarcts are common in heart cases. They are usually of the white variety, presenting yellowish-white wedge-shaped areas with the base at the surface of the cortex, and generally with a surrounding zone of hyperæmia.

Splenic infarcts often assume considerable dimensions. Various gradations between the white and the red occur. Commonly they form large wedge-shaped but sometimes irregular yellowish white areas surrounded by a zone of congestion.

Obstruction of the cerebral vessels leading to infarction of the brain, as it may be considered, usually produces a simple, white softening or encephalomalacia; but in some cases, as for example in malignant endocarditis, there may be red softening, or even such copious hæmorrhage as to obliterate all evidence of the producing embolus.

Infarction of the myocardium may itself occur from embolism of the coronary vessels. It leads to a condition of cardiomalacia, but occasionally a distinct "hæmorrhagic infarct" is produced.

In heart cases it is by no means uncommon for the pathologist to discover depressed, puckered cicatrices and remnants of scar tissue in the spleen, kidneys, and lungs, evidencing the occurrence of previous infarction.

Reference may here be made to the fact that in most cases of malignant endocarditis the resulting infarcts are usually simple, presenting no tendency to undergo suppuration, although in some of the pyæmic varieties distinct abscesses result.

Hæmorrhages are of common occurrence in heart disease. Usually they are associated with states of passive hyperæmia, embolism, or infarction.

Numerous minute cutaneous hæmorrhages are frequently met with in the final stages of cardiac affection, being particularly common about the wrists and back of the hands, but especially met with in the lower extremities.

In malignant endocarditis hæmorrhages into the skin and mucous membranes are of common occurrence.

Such forms of hæmorrhage as epistaxis, hæmoptysis, hæmatemesis, apoplexy, hæmaturia, and metrorrhagia will be best dealt with clinically.

Altered Blood States.—From what has been said regarding the secondary anæmia and hyperæmia occurring in heart disease it will be manifest that profound changes must occur in the blood.

In malignant endocarditis leucocytosis is usually well marked, and may prove of diagnostic service.

In cyanosis the blood is visibly darker than normal. Its density is increased. The hæmoglobin is raised in amount, and both forms of corpuscles increased. This increase is said to occur in congenital as well as acquired forms, and in both primary and secondary cardiac failure. Such changes are apparently not to be explained by a mere concentration of the blood arising from transudation of its more watery parts, neither is it altogether compensatory to the cyanosis, but are due probably, as Gibson suggests, to the fact that the functions of the corpuscles being lessened the tear and wear which they undergo is reduced, and the duration of their individual existence prolonged. It is, however, not clear if the condition of the blood in the superficial and more accessible parts may not vary from that in the deeper regions of the body.

Jaundice is a common condition in advanced stages of heart disease. It is generally indicative of the degree of engorgement of the liver, but may arise from an intercurrent attack of gastro-intestinal catarrh, which is by no means rare in cardiac affections. In some cases of malignant endocarditis well-marked icterus appears, and very intense jaundice is often seen towards the end of mitral cases.

Toxæmia occurs in the final stage of some few heart cases, particularly malignant endocarditis; but in the greater number of forms of cardiac lesion there is not much evidence of toxic involvement unless CO_2 be included.

Nutritional changes are specially noticeable in congenital heart disease, or lesions established early in life, "clubbed fingers" particularly becoming very marked. In a few cases of aortic incompetence failure of nutrition occurs for which there is no obvious cause.

Rupture of the heart, although sometimes described as "spontaneous," is probably in most if not in all cases to be looked upon as a terminal result of antecedent disease. Usually lesions of the coronary vessels are present, but these and the changes observed in the myocardium are referred to elsewhere.

Sudden death is one of the most striking of the pathological and clinical effects of cardiac disease. They are, however, not usually so closely connected as is generally considered to be the case. It is rare for death to occur without some evidence of cardiac embarrassment having been present. In not a few cases, however, the final stages may be rapidly passed through. More usually cardiac failure is progressive, and takes time for its evolution. In valvular affections sudden death is peculiarly associated with aortic incompetence. We have also met with several instances of sudden cardiac failure in aortic stenosis, a lesion not usually supposed to lead to such result. In mitral affections death is generally slow. In some cases of muscle failure, such as are so common in alcoholics, sudden death is by no means infrequent. Degenerate conditions of the myocardium, as in the notorious "fatty heart," and disease of the coronary arteries also frequently lead to a like termination, in the latter case angina pectoris supervening.

A consideration of the intrinsic effects of cardiac lesions, such as hypertrophy, dilatation, and other changes involving the myocardium and endocardium, may be best left until the more important individual lesions are described. The mechanism of compensation will be more clearly understood after the various special valvular lesions have been dealt with.

B. SPECIAL PATHOLOGY

Having now completed our general survey of the etiological factors of heart disease, and considered the morbid processes and their effects, it becomes necessary to describe the individual lesions met with in the more important forms of cardiac disease.

I. ENDOCARDITIS

Definition.—An inflammatory process involving the internal or lining membrane of the heart, and especially the valves.

General Etiology.—Considerable uncertainty exists as to the relative importance to be attached to the different causal factors, and hence a perfectly satisfactory etiological grouping is not at present available. Manifestly the irritant must in all cases reach the endocardium by means of the blood. In most forms of endocarditis micro-organisms have been discovered, and many pathologists claim that all varieties are of microbic origin. The so-called "malignant" cases are undoubtedly due to parasitic invasion, and hence to them the term "infection" is strictly appropriate. The exact cause, however, of the "simple" or "benign" forms, such as are more particularly associated with acute rheumatism, still remains obscure, and is likely to continue so until the true nature of the rheumatic excitant is demonstrated. Some pathologists hold that while "malignant" or "infective" endocarditis is due to virulent micro-organisms, "simple" or "rheumatic" endocarditis results from the action of attenuated forms. However this may be, certainly both pathological and clinical gradations can be traced in the forms of endocarditis, and there is something in favour of the view that the varying lesions really represent different degrees of intensity of essentially the same process. But it is quite possible that certain toxins, including the more or less hypothetical "rheumatic" toxin, are capable of initiating the morbid condition. It will thus be manifest how desirable it is at present to lay aside hard and sharp definitions, and to use our available groupings mainly as means whereby a more intimate knowledge of the different etiological factors may be attained, and a more perfect classification rendered possible.

Classification.—It has long been customary to distinguish (1) simple or benign from (2) malignant endocarditis, and on clinical as well as pathological grounds such division may well be retained, and in accordance with the same we shall consider the two varieties separately.

Other classifications are also adopted:—1. According to the severity and duration—(i.) acute, (ii.) subacute, and (iii.) chronic. 2. Dependent upon localisation—(i.) valvular and (ii.) mural. 3. When the lesion appears originally strictly local, and there is no evidence of any channel of infection, it is conveniently spoken of as—(i.) primary or protopathic, while in the majority of cases it is manifestly (ii.) secondary or deuteropathic. 4. According to the character of the lesion cases are divided into (i.) warty or verrucose, (ii.) polypous or villous, (iii.) ulcerative, and (iv.) suppurative. Combined forms are also frequent. 5. Judged by the tendencies and consequences of the process, the endocardites have been divided into (i.) necrotic or destructive; (ii.) proliferative, formative, or sclerosing; and (iii.) adhesive.

SIMPLE ENDOCARDITIS

SYN.—Benign, rheumatic, papillary endocarditis.

Simple endocarditis is met with in varying degrees of severity, and (i.) acute, (ii.) subacute, and (iii.) chronic varieties are usually described. A consideration of the chronic form may be best deferred until valvular diseases are dealt with. The acute simple variety alone need be mentioned here.

Etiology.—At present it is impossible to clearly distinguish between exciting and merely predisposing influences. While several causes are usually suggested, one causal agent stands out as pre-eminent—rheumatism. Many consider it as sometimes a primary rheumatic manifestation. Certainly endocarditis occurs in association with acute polyarticular rheumatism, or "rheumatic fever," in proportions variously estimated as from 20 to 80 per cent. The younger the subject the more common the association. It is sometimes said that the more violent the articular rheumatism, the greater the coincidence of endocarditis, but in a large number of cases it develops in association with very mild or even unrecognised attacks of rheumatism. The first attack of rheumatic fever usually proves more damaging than later ones. Usually the endocarditis commences early in the course of the rheumatic attack, although the condition may be undetected at the time.

Sometimes endocarditis develops during or follows less manifest rheumatic processes. The endocarditis occurring in scarlatina is sometimes, and probably in gonorrhœal rheumatism always, of an "infective" type. Endocarditis is rare in monarticular rheumatism and in chronic rheumatism.

Amongst 29 cases submitted to autopsy with apparently acute "simple" endocarditis, in the Pathological Department of the Manchester Royal Infirmary, a clear history of rheumatism had been obtained in 17, and there was reason to believe that rheumatic manifestations had been present in even a larger number. In 20 there was also evidence of old-standing valvular disease.

Whether or not chorea is a manifestation of rheumatism as some would believe, it is at all events commonly associated with rheumatism and endocarditis. In each of 4 cases recently submitted to autopsy at the

Manchester Royal Infirmary, vegetations covered both mitral and aortic valves. Probably some degree of endocarditis occurs in from 40 to 60 per cent of all cases of chorea.

Endocarditis occasionally occurs in connection with the infectious fevers, particularly scarlet fever. Quite exceptionally endocarditis arises in the course of other acute infectious diseases, such as measles, small-pox, diphtheria, typhoid, and malaria, and is possibly then truly of "infective" type.

It is also said that acute endocarditis has in some few instances followed injury to the chest or rupture of a valve by strain.

Among the predisposing conditions reference may be made to the following:—In young subjects acute endocarditis occurs with somewhat greater frequency in females. In later life the difference of sex is less marked. It is most common in young subjects and early adult life. It is exceedingly rare in old people, a first attack being then practically unknown. The average age of fatal Manchester cases of acute endocarditis was a little over 26 years. Pre-natal endocarditis undoubtedly occurs; indeed some pathologists consider fœtal endocarditis as by no means rare. Endocarditis is not infrequently associated with malformations, particularly of the valves.

Transmitted influences are of importance, apparently only in so far as they lead to the development of rheumatism.

Conditions of climate, social position, occupation and habit, are probably only of moment in so far as they expose the subject to the influence of the exciting agents of endocarditis, particularly rheumatism.

Morbid Anatomy.—The lesions met with in acute endocarditis are usually most conspicuous in connection with the valves, and principally affect those of the left side of the heart, except in fœtal life. The mitral valve is most commonly involved, but in fatal cases vegetations are usually found on both aortic and mitral segments. The vegetations usually appear as a row or fringe of bead-like prominences on the auricular aspect of the auriculo-ventricular cusps, and on the ventricular aspect of the sigmoid segments, but situated a little distance from the free edge along a line corresponding to the points of maximum contact. Sometimes the vegetations are large and assume pedunculated or villous-like forms. The development of such large fungating masses is a more characteristic feature of malignant endocarditis. Sometimes a few vegetations will be found on the chordæ tendineæ, but in the simple form of endocarditis they do not generally extend to the mural endocardium.

In fatal cases care must be taken to carefully distinguish between (i.) the lesions depending on the endocarditis, and (ii.) those merely associated with the endocarditis.

Acute pericarditis is common. Inflammatory and degenerative myocardial changes are frequent, and probably account for the rapid dilatation which occasionally leads to a fatal issue. Infarction is much less common than in the malignant form. Adherent pericardium is not infrequently met with in fatal cases, generally showing that the fatal attack has been preceded by former similar attacks.

The effects of simple endocarditis may be briefly considered. They may be (i.) local and (ii.) general.

Resolution with absorption of the inflammatory products and more or less complete repair of the affected portion of the endocardium very probably occurs in a number of the milder cases.

Adhesion, cicatrisation, and induration result in a considerable number leading to valvular disease.

Valvular aneurysms occasionally are formed, but are much more likely to occur in the "infective" forms.

Rupture of a valve or chordæ tendineæ is only of exceptional occurrence.

Local myocarditis and fibrosis of the musculi papillares form far-reaching consequences.

Secondary infection leading to the development of a "malignant" endocarditis is a possibility ever to be borne in mind.

Anæmia when it occurs is probably due to the influence of the rheumatic "toxin." It possibly predisposes to the development of infective endocarditis.

Embolism is comparatively rare in simple endocarditis, while in infective endocarditis, and in chronic valvular disease, it is of frequent occurrence.

Morbid Histology.—The minute changes consist in a proliferation of the endothelial cells; multiplication of the connective tissue cells of the subendocardial layer; infiltration with leucocytes, serum, and fibrin; and deposition of fibrin and blood-clot on the free surface from the circulating blood. More or less localised myocarditis is apt to occur, with inflammatory infiltration of the inter-muscular tissue and lymph spaces.

Micro-organisms are sometimes found in the superficial parts of the vegetations, but usually not in the deeper structures. Many hold that this is evidence that the organisms are not to be considered as etiological agents, but rather dependent on a "terminal" infection.

SUBACUTE ENDOCARDITIS

This form scarcely calls for special description. Both simple and malignant forms may run a subacute course. Associated with the proliferative process are more or less well-marked evidences of conservative efforts characterised usually by the formation of fibrous tissue and resulting in a certain degree of deformity of the affected parts. Adhesions between the cusps and chordæ tendineæ are liable to occur. The condition readily passes into a chronic stage producing conspicuous valvular deficiencies.

MALIGNANT ENDOCARDITIS

SYN.—Arterial pyæmia, infective, mycotic, septic or ulcerative endocarditis.

Definition.—A process resulting from the invasion of the endocardium by micro-organisms, and characterised by proliferative, destructive, or suppurative changes.

Etiology.—In pre-bacillary days this form of endocarditis was recognised, but it remained for the bacteriological investigations of recent years to clearly demonstrate its dependence on microbic infection.

Although always the outcome of microbial invasion, the process cannot, however, be considered a distinct pathological entity, for it probably results from infection by several distinct species of organisms.

In some instances the involvement of the endocardium appears as (i.) a primary lesion; but in perhaps the majority it is found to be (ii.) secondary, either to some other infective process occurring within the body, or having some channel of introduction whence the infective organisms can be definitely traced, as from wounds or septic foci.

Thus three classes may roughly be distinguished:—

i. Where there is no demonstrable source of infection or evidence as to the channel of microbial entrance.
ii. Where there is an evident source of infection.
iii. Where the process is associated with other infections, as some of the infectious fevers, pneumonia, diphtheria, typhoid, tuberculosis, and the like.

The organisms most frequently met with (i.) belong to the pyogenic group, and include:—

Streptococcus pyogenes.
Staphylococcus pyogenes albus.
Staphylococcus pyogenes aureus.
Bacillus pyogenes fœtidus.
Bacillus coli communis.

ii. Organisms occurring in connection with other diseases occasionally play an important part, and the following have been detected in the vegetations and ulcerations :—

Diplococcus pneumoniæ.
Pneumobacillus of Friedländer.
Tubercle bacillus.
Typhoid bacillus.
Bacillus of diphtheria.
Bacillus of influenza.
Gonococcus.

iii. Some few organisms not as yet known to be associated with any other infective process have also been described :—

Bacillus endocarditis griseus.
Micrococcus endocarditis rugatus.
Micrococcus zymogenes.
Bacillus endocarditis capsulatus.
Bacillus immobiles et fœtidus.

It is certainly at present difficult, if not impossible, to apportion the relative importance of the different organisms in initiating and maintaining the morbid process. In some instances the condition is apparently the direct result of some definite microbe, and where such can be proved to be the case, the term "infective" should be replaced by that of the specific agent, *i.e.* streptococcal endocarditis, gonococcal endocarditis.

In not a few cases, however, the condition would appear to be the outcome of a mixed infection; at all events, either by later, or terminal, or post-mortem invasion, more than one form of microbe gains access. But this is only in accordance with what is known to occur elsewhere in connection with infective processes.

Unfortunately, examination of the blood during life does not usually afford positive results.

The experimental establishment of endocarditis need not be dwelt on here; suffice it to say that investigations on the lower animals—rabbits and dogs—have clearly demonstrated the possibility of developing a condition practically identical with the malignant endocarditis of man.

But there are important predisposing influences. Malignant endocarditis is commonly found to be engrafted on a chronic inflammatory or degenerative condition of the valves. Sometimes a malformation, possibly due to fœtal endocarditis, has been noted. In some few instances an imperfect ventricular septum has been found. In an analysis of 65 fatal cases of malignant endocarditis occurring in the Manchester Royal Infirmary of recent years, of whom not quite 34 per cent gave a clear history of rheumatism, in no less than 50 or 77 per cent was there distinct evidence of old endocardial mischief. In 2 there was adherent pericardium; mitral stenosis existed in 9; aortic stenosis in 2. In one there was congenital malformation. In another the aorta was narrowed and thickened. In several the affected valves were also the seat of calcareous deposition. In only 12 of the cases were there fairly reliable grounds for believing that the valves had been normal previous to the development of the malignant endocarditis.

Occasionally in the course of acute polyarticular rheumatism malignant endocarditis develops. Usually, however, rheumatic fever gives rise to the simple or benign form, and it is quite possible that the "rheumatic" pains complained of in some infective cases should be looked upon as of a septic nature.

Chorea also, although quite exceptionally, is followed by malignant endocarditis.

As already indicated, the organisms of not a few of the general infectious diseases are capable, under certain circumstances, of acting as special pathogenic excitants to the cardiac valves. Chief amongst these may be enumerated pyæmia, erysipelas, pneumonia, typhoid, diphtheria, scarlet fever, influenza, dysentery, and malaria.

Malignant endocarditis sometimes follows parturition, when it may be considered as secondary to a puerperal septic infection.

Suppurative conditions may lead to this form of cardiac involvement. Pyogenic organisms not infrequently gain access to the blood-stream from a local collection of pus or from suppurating wounds. Thus such conditions as empyema, local purulent peritonitis, otitis media, suppurative meningitis, suppurative cholecystitis, have been followed by malignant endocarditis.

It must not be forgotten that when the case comes under observation, no trace of the channel of infection may be detected, and the patient may make no mention of one ever having existed.

Traumatism in the form of injuries to the external surfaces of the body, or internal surface membranes, may become of etiological importance, in so far as thereby channels for the invasion of organisms are established. There is also reason to believe that blows on the chest occasionally lead to the development of an infective endocarditis, possibly by producing some minute injury to the valve segments or endocardium, and so affording access to organisms which may be present in the blood.

Season and climate have been thought to be of some moment. It has been suggested that malignant endocarditis occurs most frequently in the autumn months, but this is very doubtful. In some parts of the country it seems to be much more frequently met with than in others. In Manchester and district, where the disease is only too common, we have known three autopsies on cases to be made in one week. Some have attempted to show that the affection was connected with decaying vegetation. Malaria probably does not do more than predispose. It is reasonable to believe that all conditions leading to deterioration of health should be credited as being more or less influential as predisposing factors.

Men are considerably more liable than women. Of 65 fatal Manchester cases 46 were men, 15 women, 3 boys, and 1 girl; that is, 49 males and 16 females, or a percentage of a little over 75 for men and nearly 25 for women.

Malignant endocarditis is essentially a disease of adult life. The large majority of cases are met with between the ages of 20 and 40. In our Manchester cases the average age was 30½ years. The youngest was 6; the oldest 72 years.

Morbid Anatomy.—The extent and character of the lesions met with in the heart vary considerably. An attempt has been made to recognise varieties according to the predominant feature (i.) vegetative, (ii.) ulcerative, and (iii.) suppurative, but such a division does little more than indicate stages, and all conditions may be met with in the same case.

The valves are more particularly affected, but the chordæ tendineæ and parietal endocardium are frequently involved, and the process sometimes extends into the aorta. The left side of the heart is generally affected, although the tricuspid and pulmonary valves may also be involved.

Of our 65 Manchester cases, in only one instance was the malignant endocarditis limited to the right side. In 5 both right and left cavities were involved, the tricuspid and mitral in 2, and the tricuspid, mitral, and aortic in 2. In only one case were the pulmonary valves affected, and their vegetations were found in the pulmonary artery and right ventricle. In 17 the mitral segments were alone involved, in 18 the aortic. In 20 both aortic and mitral valves were the seat of vegetations. In a considerable number the process had extended from the valves and chordæ tendineæ to the adjacent parts. In several the wall of the left auricle was involved. In 4 cases vegetations were present in the aorta itself. In one they extended into the coronary artery. In another they surrounded the opening of one of the coronary vessels. As to the character of the lesions, in 25 there was extensive formation of vegetation, in 34 more or less ulceration. In one there was local suppuration, and in another the mitral valve was the seat of an acute aneurysm. In at least one instance there was apparently recent calcareous deposition in the vegetations.

The vegetations vary greatly in size and extent. Sometimes they are small and scanty, frequently they form large, irregular, pedunculated, shaggy masses. They are generally of a gray or pinkish-yellow colour, and often covered with blood-clot. Sometimes, as already indicated, they are the seat of a recent calcareous infiltration.

The destructive character of the process is commonly indicated by the presence of ulceration. Sometimes the ulcers are small and superficial; more frequently they are large and deep, penetrating through the valves, extending to the chordæ tendineæ, which are frequently eroded and ruptured, and sometimes penetrating deeply into the myocardium.

The vegetations are usually very irregular in arrangement, contrasting greatly with the more or less limited distribution of those in simple endocarditis. They frequently extend to the mitral endocardium, and particularly is this the case when the chordæ tendineæ of the anterior flap of the mitral are ulcerated through, abundant vegetation being then usually present on the posterior and outer part of the wall of the left auricle. Occasionally the vegetations extend upwards along the aorta and pulmonary artery, and sometimes encroach upon the orifices, and even spread into the channels of the coronary vessels.

Mural malignant endocarditis without involvement of the valves is almost unknown. Occasionally an aneurysm of a valve is produced. Sometimes foci of suppuration occur in the valves, or small abscesses may form in the subendocardial and myocardial tissues. In this way cardiac aneurysm or even penetration and rupture of the heart may result.

Secondary and Associated Lesions.—But the lesions noted in cases of malignant endocarditis must be carefully distinguished accordingly as they (i.) are primary and have preceded the endocarditis, or are (ii.) secondary and due to or associated with it. In the latter cases they will usually be the result of (1) embolism or (2) toxic absorption.

The secondary lesions include infarction of the lungs, spleen, and kidneys; cerebral embolism and softening; embolic blocking of mesenteric vessels or peripheral arteries; and multiple hæmorrhages into the skin, serous membranes and retina.

Congestive and degenerative states of various organs are common. The spleen is usually much enlarged. The kidneys also frequently present profound tubular changes. Infarcts manifest much caprice in their distribution. It is, however, exceptional for the secondary embolic centres to become the seat of suppuration.

Reference to some of the conditions noted in the 65 fatal Manchester cases may not be without interest. Infarction occurred in 34. The spleen was involved in 20, the kidneys in 11, the lungs in 7. In one instance an embolus blocked the right middle cerebral, in another the femoral, and once an embolic aneurysm of the coronary artery was found. Acute lobar pneumonia existed in 12. In the majority of these it was apparently secondary, and possibly in several of embolic origin. Hæmorrhage into the lungs existed in a number. Acute pleurisy was marked in 7, and in 3 empyema had occurred. Acute pulmonary tuberculosis, with extensive excavation and tuberculous ulceration of the intestines, was met with once. Acute pericarditis was found in 4, and old adherent pericardium also in 4. Peritonitis was present in 3, one of these being evidently tuberculous. Gastric ulcer of recent formation occurred once. In two instances a duodenal ulcer had led to death from hæmorrhage. In another case intestinal ulceration existed, possibly of embolic origin. Suppurative cholecystitis was found twice; in one the gall-bladder was obstructed by old inflammatory thickening, but no calculi were present. Hepatic abscesses were noted once, and in the same case there was extensive abdominal tuberculosis. Well-defined hepatic cirrhosis was present in at least 4. Most cases had a more or less "nutmeg" condition of the liver. Splenic enlargement was a special feature in 53. In another case which had appeared during life to be one of "splenic anæmia," though there had been prolonged intermittent pyrexia, the spleen was of enormous size. The average weight in 55 cases was a little over 16 oz. The great variation in size and weight will be seen from the following figures:—One weighed 70 oz., 2 over 30 oz., 15 between 20 oz. and 30 oz., 24 between 10 oz. and 20 oz. In a few instances the spleen was not appreciably enlarged post-mortem. Nephritis mainly of a parenchymatous type existed in 11 cases. "Granular" kidney was met with once. Acute septic meningitis was noted twice. In another case puriform fluid was found within the lateral ventricles. Cerebral softening existed in 3, in 2 being manifestly of embolic origin. Meningeal hæmorrhages occurred twice. Extensive cerebral hæmorrhages were present in 3, and in only one could a distinct embolus be found. Embolism of the femoral occurred once. Pus in the joints was present twice, and boils were also noted in 2 cases.

Morbid Histology.—The action of the micro-organisms leads to changes which are partly (i.) necrotic, but in other portions (ii.) inflammatory.

Where the endocardium is far removed from vessels, as in the greater part of the valves, unless sclerosed, the former process is predominant, but where the microbic invasion occurs in proximity to vascular regions an inflammatory exudation speedily follows.

The action of the organisms or their toxins leads at first to rapid degeneration and later to molecular death of the infected tissues, and the chemico-physical changes in the affected part produce clotting over the diseased structures. In vascular regions extensive inflammatory exudation occurs. The leucocytes are collected sometimes in sufficient numbers to constitute distinct areas of suppuration. In less acute cases proliferation of the connective tissue elements occurs. The endothelium and portions of the necrosed tissue are readily detached, and the projecting cellular elements becoming the seat of deposits of fibrin form irregular, soft vegetations. By the separation of the dead tissue and the detachment of the vegetations ulcers are produced. Ulceration and the formation of vegetations are usually met with in association.

Suitable staining reagents demonstrate the presence of the infecting micro-organisms in the affected parts.

Post-mortem examination of these cases should include a full and complete bacteriological investigation of the blood, vegetations, splenic pulp, and secondary foci when present. Every care must be taken to prevent any extraneous contamination. Particulars as to technique will be found in the ordinary works on bacteriology.

Pathological Chemistry.—Recent researches have shown that the products of the growth of certain organisms taken from cases of infective endocarditis are of the nature, the one of a proteid consisting of proto-albumose and deutero-albumose, and the other, a non-proteid body with strong acid reaction.

II. VALVULAR AFFECTIONS

Before considering the various morbid conditions producing inefficiency of the individual valves, reference may be made to certain general pathological features; for it is only by the painstaking comparison of clinical phenomena with the results of pathological investigation, that mere vague generalisations or inaccurate deductions can be successfully overturned.

Etiology.—The precise nature of the influences predisposing to or exciting valvular disease is in only too many instances far from clear. In some the morbific influence operates during intra-uterine development.

The agencies producing valvular disease are frequently so combined as to render a strict differentiation into predisposing and exciting influences impossible. It would seem that in many instances a distinctly extraneous agent can only exert its pathogenic power when assisted by intrinsic conditions tending to lower the general or local resistance of the body, or to increase the physiological work of the part affected. Undoubtedly infective agents hold an important position as excitants, and there is every reason to believe that toxic bodies, either of autogenetic or heterogenetic origin, play an important part in the etiology of valvular disease of the heart.

Heredity.—Family transmission of a tendency to valvular disease may be admitted as a possibility, but acting probably by the continuance of constitutional disease or hereditary depravity of tissue. The importance of the vascular system, and with it, of necessity, the central organ in relation to heredity, cannot be doubted. The family connection of coronary disease and angina pectoris is clearly established. States of high arterial tension also seem to "run in families," and several children of the same parents may suffer from an apparently similar form of cardiac inadequacy. Race, as far as is known, is not of particular etiological importance.

Sex plays a determining part, as indicated by the fact that mitral stenosis is commonest in women, while aortic disease is most frequent in men. This influence of sex would seem to be mainly dependent on exposure to exciting conditions; thus rheumatic affections are undoubtedly most usually met with in females; while males, by their occupation or exposure to agencies producing arterial and muscular degenerative changes, are most liable to valvular defects from an aortitis implicating the function of the valves in one way or another.

Age.—Valvular defects, as already indicated, may originate during intra-uterine life. During the period of normal development, childhood, and early adult life, morbid processes of an inflammatory nature, and usually rheumatic in origin, are particularly prone to affect the valves. Microbic valvulitis, or an endocarditis dependent on invasion by infective organisms, may occur at any age, but is most frequent in adults. Auriculo-ventricular valvular defects arising from primary muscle failure are usually met with in mature subjects, and generally at what should be the period of maximum vigour. In advanced and declining years the aortic valves are peculiarly prone to suffer from degenerative changes, and are liable to be involved by low inflammatory and degradation processes extending to them from adjacent parts. Thickening of the auriculo-ventricular segments is not infrequently met with by the pathologist when there had been no clinical evidence of impairment of their action. In females the period of sexual activity, with its elevations of arterial tension and the strain of child-bearing, is of considerable importance in advancing a valvular condition practically latent to one associated with marked symptoms.

Temperament is a factor which may be considered of some slight etiological importance in accelerating or retarding the day of cardiac failure. The high tension engendered by worry is apt to be neglected.

Previous Disease.—As already indicated, a valve in any way impaired, either by congenital deficiencies or acquired defects, is prone to become the seat of further morbid processes, and this would seem to be particularly the case when the exciting agent is of an infectious nature.

Climate.—The manifold factors included under this term can only be considered of importance in so far as they influence the production of diseases, like rheumatism, which are intimately allied with morbid processes occurring in the valves. The relation of Bright's disease to climate must also be noted, for the connection of renal affections and certain forms of cardiac disease is a close one.

Social position becomes of importance in influencing exposure to definite morbific influences. The inability of the poor to obtain a livelihood without hard work means early failure to many a cardiac cripple.

Occupation must be admitted as not only a predisposing factor, but oftentimes a contributing influence. The importance of work necessitating stress and strain cannot be set aside. Its action is complex: sometimes it may act through sudden injury, but more frequently it leads to valvular deficiencies by the establishment of chronic inflammatory processes, causing aortic dilatation or direct implication of the valves, while there may be at the same time muscle failure brought about by coronary interference or otherwise.

Education, or rather its associated methods, unfortunately cannot at present be excluded from the group of predisposing causes. There is reason to believe that mal-education, at least in physical procedures, oftentimes precipitates a cardiac breakdown in young subjects, who, perhaps, with slight valvular impairment following rheumatic endocarditis, are quite unfitted for specially trying forms of drill, exercise, or athletic pursuits. Still, taken altogether, the influence of athletics is beneficial; the good outweighs the evil.

Habits are of importance, as, for example, alcoholism, sexual excesses, depraved feeding, etc.; but reference to these will be found in the clinical section.

Exciting Causes.—Among the specific influences productive of valvular disease *rheumatism* stands out pre-eminent. As to its precise nature we are still in the dark. We may safely assert, however, that by the prevention of rheumatic fever, and such "rheumatic" manifestations as chorea, rheumatic arthritis, and "growing pains," the greater number of chronic valvular affections would be effectually banished. In every case of chronic valvular disease a history of rheumatism, or the so-called "rheumatic" affections, should be sought. Several of the infectious diseases are said occasionally to give rise to progressive valvular endocarditis. Possibly scarlet fever may sometimes so act; but even here the relation to rheumatism must not be lost sight of, for there are several quite distinct forms of arthritis met with in scarlatina. Septic and other organisms, as previously indicated, may establish or continue valvular disease, and usually such proceed more or less rapidly to a fatal issue; but some pathologists contend that death of the microbes and subsequent cicatrisation of the valves occasionally occur. Such a view is exceedingly difficult, if not impossible, to prove.

Both autotoxic and heterotoxic agents seem capable of initiating chronic inflammatory and degenerative changes in the fibrous and muscular elements of the heart, and in the effects of which the valves may possibly participate. Syphilis, gout, and Bright's disease must here be mentioned. Syphilis, however, although acting disastrously on the coats of the aorta, so leading to dilatation and valvular incompetence, does not commonly affect the valves themselves. One form of localised supra-valvular aortitis, of which we have met with several examples, is probably syphilitic in origin, and has a special tendency to produce obstruction of the coronary arteries and extension to the aortic valves, rendering them incompetent. Gout, alcohol, and Bright's disease probably never give rise to valvular disease directly. Gout, and with it plumbism and Bright's disease, are of course associated with high arterial tension and implication of the vascular system, and thence secondary valve implication or incompetence may arise.

The etiological importance of mechanical strain has already been insisted upon, and direct or indirect violence must also be admitted as a possible, although, under normal circumstances, most unlikely cause of valvular deficiency.

Morbid Anatomy.—A clear comprehension of the gross lesions affecting the valves of the heart will do much to simplify the clinical manifestations, and to elucidate the consequences and associated changes of valvular disease.

As already indicated, the valves of the left chamber of the heart are most commonly affected. It is usually only in fœtal and infective endocarditis that the right side is alone involved. In simple as well as in malignant endocarditis both sides of the heart may suffer, but even then the involvement of the left is usually most extensive. Double auriculo-ventricular stenosis is not very rare, but when met with the left-sided lesion is always far in advance of that of the right.

The valves may suffer primarily, or become impaired only secondarily. The exact mechanical deficiency predominant may be more or less of an accident pathologically speaking, and between the huge orifice of a highly incompetent mitral orifice and the chink-like lumen of an extreme stenosis almost every gradation may occur.

Stenosis of an orifice is produced by the thickening and contraction of the valve segments, by their adherence to each other, to the thickening, shortening, and fusion of adjacent chordæ tendineæ, by deposition of lime salts in the structure of the valve increasing its rigidity, preventing close apposition of segments, and forming a narrow yet possibly ever open door.

Occasionally aneurysmal dilatation of the valves also produces some degree of obstruction, but this is not so frequently the case as in acute endocarditis, and the condition is rather a pathological curiosity than a fact of practical importance.

Incompetence may arise from defects in the valves themselves such as above indicated, or may be secondary to deficient systolic narrowing of the orifice because of muscle failure; or inefficiency of contraction of the musculi papillares may interfere with the control normally given to the valves by the chordæ tendineæ. Indeed, in considering mitral lesions it is impossible to eliminate the effects of associated muscle failure from the causes at work in the production of the valvular incompetence.

Morbid Histology.—The essential features of chronic disease of the valves consist in the production of a fibrous cicatricial-like tissue, in which calcareous salts are peculiarly prone to be deposited.

Consequences and Associated Changes.—The effects of valvular disease may be divided into (i.) intra-cardiac and (ii.) extra-cardiac. It is only necessary to mention them here as they are referred to elsewhere.

1. Alteration in the size, weight, shape, and relative proportions of the different parts of the heart.
2. Extension of sclerosis to the adjacent endocardium and formation of localised thickenings and irregularities.
3. In some instances diminished blood-supply, with consequent anæmia and impaired nutrition.
4. Disturbances of the pulmonary circulation.
5. Distension of the venæ cavæ and their tributaries.
6. Intra-cardiac clotting with formation of cardiac thrombi.
7. Formation of emboli.
8. Hæmorrhage from simple engorgement of vessels apart from embolism.
9. Dropsy.
10. Blood-changes.

AFFECTIONS OF THE AORTIC VALVES AND ORIFICE

Aortic disease dependent on valvular lesion may occur:

1. By primary involvement.

- (i.) Of acute development.
  - (*a*) External violence.
  - (*b*) Rupture.
  - (*c*) Malignant endocarditis.
- (ii.) Of slow development.
  - (*a*) Maldevelopment.
  - (*b*) Chronic valvular endocarditis.
  - (*c*) Degradation processes.

2. By secondary involvement.
   - (i.) Of acute development.
     - (*a*) Extension of acute aortitis.
     - (*b*) Extension of malignant endocarditis.
   - (ii.) Of slow development.
     - (*a*) Extension of chronic aortitis.
     - (*b*) Extension of chronic endocarditis.

Etiology.—Disease of the aortic region of the heart impairing the efficiency of the valves is essentially an affection of adult men, although, of course, it is not infrequently met with in young subjects of both sexes in the form of an infective endocarditis, or as a consequence of a rheumatic affection.

It is the aortic valves which are most markedly exposed to and deteriorated by the stress and strain of excessive or unregulated physical exertion. Under such circumstances disease of the aorta has generally preceded the crippling of the valve segments, and having accomplished their incompetence by causing dilatation of the vessel by virtue of impairing its elasticity, and throwing strain upon it during the ventricular systole. A rheumatic affection of the aortic valves is usually met with in association with involvement of the mitral segments, but in a certain number of instances the aortic valves are the only ones seriously impaired. In the production of the crippling sclerosis which accounts for such a large number of cases of aortic deficiency in addition to strain, gout and Bright's disease, are often said to be of etiological importance, but they are so probably only through the influence of vascular pathological states. Certainly sclerotic valves are commonly met with in subjects who have never experienced the toxic influences of gout nor suffered from Bright's disease, while on the other hand chronic endocarditis probably never occurs as a direct consequence of these states. Malaria has been credited with being instrumental in establishing a chronic aortitis. All conditions inducing high arterial tension must be considered as agencies making for strain of the aortic valves. Such occupations as those of miner, collier, smith, soldier, and the like, undoubtedly predispose to aortic disease. Syphilis, however, must also be admitted as a factor in some of these cases.

External violence and strain may, under exceptional circumstances, lead to sudden rupture of an aortic valve. In some of the recorded cases there is reason to believe tearing occurred in a valve already weakened by existing disease. The valvular laceration occurs during severe muscular exertion, and is immediately followed by more or less cardiac pain and distress, and usually marked dyspnœa.

Extensive mechanical defects often result from malignant endocarditis. The abundant flocculent vegetations may tend to occlude the orifice, or the formation of a valvular aneurysm may produce a degree of obstruction, but more frequently extensive ulceration leads to a rapidly established incompetence permitting of free regurgitation.

Maldevelopment or disease during intra-uterine life is a rare cause of aortic disease.

It is well to note that it is by no means rare to find a thin fringe of fine bead-like vegetations in a rheumatic case along the ventricular aspect of the cusps, which during life had presented no clinical evidences of mechanical deficiency.

Chronic aortitis is one of the most prolific agencies by which aortic valvular disease is produced. From the clinical point of view the actual form of aortitis is of comparatively little importance. Most frequently it is of an atheromatous variety. The aortitis deformans may lead to incompetence without directly involving the valve segments by producing dilatation of the aorta, and ultimately an increase in the size of the aortic ring, but often there is also stiffening and thickening of the cusps.

Reference may here be made to a special form of aortic disease leading to a certain degree of incompetence, and usually producing considerable impairment of the cardiac nutrition by narrowing the orifices of the coronary arteries. The condition involves most markedly the aorta immediately above the valves. It consists of a chronic localised aortitis, and occurs usually in patches, the borders having often an undulating or serpiginous outline very suggestive of a syphilitic process. In the cases we have examined, however, no very distinct syphilitic lesions could be detected elsewhere in the body. In the case of a young woman where this condition existed, and where angina pectoris was a prominent symptom, pelvic inflammation had existed, and there were other reasons to believe syphilitic infection had occurred.

Morbid Anatomy.—Brief reference may be made to the lesions found :—

1. *In Aortic Incompetence.* — In cases of aortic regurgitation due to chronic endocarditis and sclerosis of the valve segments, conditions are found which in most instances are the result of an association of processes. There is also oftentimes considerable variation in the seat and extent as well as in the predominant character of the morbid conditions.

In some instances the portion of the cusp nearest the base of attachment is indurated, and it may be contracted while the remaining portion is practically uninvolved. Here the normal part may be retroverted or doubled backwards towards the ventricular cavity in consequence of the failure of the segments by approximation to sustain the aortic blood-pressure during diastole.

Sometimes the segments are merely uniformly thickened. They may be irregularly thickened, puckered, and extensively deformed. Oftentimes the induration more particularly affects the free edges of the flaps, which may be rounded and everted or rolled towards the base of the aorta. Occasionally one of the cusps appears as though dragged away from its attachment, and droops ventricle-wards. Adhesions are sometimes formed between adjacent flaps, and when these are broken through a large pouch may be formed by the combined cusps. We have met with distinct aneurysmal pouching in a sclerosed valve.

In many of the cases where calcification of the valves is a conspicuous feature the aorta is practically healthy. This would seem to indicate that the valves had been affected primarily. When of rheumatic origin there is a particular disposition to the deposition of lime salts. In the past there has been too great a tendency to consider calcareous infiltration as evidence of the process loosely termed "atheroma."

Where calcification is a conspicuous feature, it is well to observe that the deposit of lime salts is often not limited to the valves, but may be abundant in the infra-valvular portion of the septum and in the anterior segment of the mitral.

Incompetence may arise from dilatation of the aortic ring without any distinct involvement of the valve segments themselves.

2. *In Aortic Stenosis.* — Chronic rheumatic endocarditis is particularly influential in leading to aortic stenosis.

Pure cases of aortic stenosis are by no means common, although a certain degree of obstruction exists in a large number of the cases in which incompetence is the conspicuous lesion.

Aortic stenosis is always due to valvular disease. The actual lesion presents considerable variation in its exact form, seat, and extent.

Sometimes the whole of the cusps are so infiltrated with lime salts as to be separately indistinguishable, and forming a calcified ring, in the centre of which is a mere chink of a lumen. Or the valves may be thickened, fused one to the other so as to constitute a kind of funnel with its narrow end or apex projecting into the aorta.

Frequently the segments are irregularly involved. In some cases where ther is abundant deposition of lime salts, intervening portions of fairly healthy valv tissue may sometimes be observed. Commonly the valves are thickened an rigid, coherent, and, according to some pathologists, rendered rough by th calcification of adherent thrombi, the lumen being often so narrow as to barel admit a goose-quill.

3. *Aortic Incompetence with Stenosis.*—From what has already been said it wil be clear from the pathological necessities of the case that mixed or combined co ditions must occur. Such is undoubtedly the case. In a considerable number

instances where incompetence is the conspicuous clinical feature some degree of aortic obstruction will be found post-mortem.

Affections of the Mitral Valves

The nature of the processes leading to deficiency of the mitral valves do not differ from those already described as occurring in valves generally, or in the aortic segments in particular. It will, therefore, only be necessary to refer to points of special practical importance. The following forms of mitral disease may be distinguished:—

1. Mitral stenosis.
2. Mitral stenosis with incompetence.
3. Mitral incompetence.

Although doubtless in most cases in some stage of a chronic primary valvular disease of the mitral segments combined mechanical defects occur, yet for purposes of practical discrimination, as well as for convenience of grouping and description, the above three classes may be recognised. It may, nevertheless, be best to add at once that in nearly all cases of mitral stenosis incompetence of a greater or less degree takes place at some period.

Mitral Stenosis.—Left auriculo-ventricular obstruction forms one of the most important of the organic diseases of the heart. It occurs in the majority of cases associated with some incompetence.

Etiology.—Mitral stenosis is particularly common in females, although met with also in a large number of males. While often not coming under medical observation till adult life, and sometimes not until the latter part of middle life, the condition is generally initiated at an early age; the largest number of cases are met with before the age of thirty. The great majority of cases are due to a chronic endocarditis. Sometimes this would seem to arise as an acute condition. Rheumatic fever or chorea are the common antecedents. In some instances no definite excitant can be detected. Where no distinct rheumatic history is forthcoming careful inquiry will frequently elicit some reference to "growing pains" or chorea.

In some very rare instances mitral obstruction has been dependent on the abundant formation of vegetations, or the intrusion of a so-called cardiac polypus or tumour.

Morbid Anatomy.—Mitral stenosis is the result of very conspicuous organic changes in the valves. The auriculo-ventricular ring and the structures in immediate connection may be involved. Occasionally the narrowing of the mitral orifice appears to be mainly, and according to some even entirely due to an induration with contraction, and sometimes calcareous deposition in the fibrous ring encircling the auriculo-ventricular orifice and the base of the valve, as well as in the immediately adjacent muscular tissue. Most usually the stenosis is due to extensive changes in the valve segments. The sclerosis and cicatricial formation is apparently the result of a chronic inflammatory process. The valve segments, and usually the anterior or aortic cusp is the one principally involved, are much thickened, of firm and oftentimes cartilaginous consistency, and frequently puckered, shrunken, or otherwise deformed. A variable degree of calcareous infiltration is of common occurrence. The chordæ tendineæ are usually greatly thickened, very rigid, shortened, and often fused or firmly adherent in their course to the valves, forming stiff ridges of dense fibrous tissue, or actually incorporated into the sclerosed funnel. The apices of the musculi papillares are frequently converted into dense white cartilaginous-like tissue. The lumen of the auriculo-ventricular orifice is of course narrowed. A normal adult mitral orifice should readily admit two finger-tips. A stenosed mitral orifice frequently will only allow of the insertion of the tip of the forefinger, and sometimes the degree of stenosis is still greater.

According to the structural deformity two forms of mitral stenosis have been described: (i.) the buttonhole form, and (ii.) the funnel-shaped variety. In the former the mitral orifice presents the appearance when viewed from its auricular aspect of a slit in a tough and rigid diaphragm. In the latter the contracted cusps, with fused chordæ tendineæ projecting ventricle-wards, gives the aspect of that of a funnel. Often, however, what looks like a button-hole variety when viewed from above will assume a more or less typical funnel-shape appearance when viewed from below. There would, therefore, seem to be no clear pathological reason or clinical advantage in insisting on this somewhat artificial division.

It is well to remember that in many cases of mitral stenosis the natural seat of the auriculo-ventricular orifice is shifted, and instead of being on a level with the base of the valve segments, is lowered to a position at the apex of the funnel-shaped rigid valve.

Still further the obstruction is occasionally increased by the formation of thrombi on the auricular surface of the sclerosed valve, or by pendulous coagula from the left auricle.

The results of this lesion on the general circulation have already been sufficiently described, and certain of the local effects will be referred to presently when considering hypertrophy and dilatation, so that it is only necessary here to mention the more conspicuous intra-cardiac consequences and associations. Naturally the chief stress of mitral stenosis falls on the left auricle, which, according to the stage of the lesion, presents variable degrees of hypertrophy and dilatation.

There is also considerable increase in the size of the right ventricle, the hypertrophy of its walls and prominence of its infundibulum being usually most conspicuous.

In the early stages hypertrophy is the chief if not sole feature, but in old-standing cases, where a certain amount of mitral incompetence has existed with progressive heart failure, more or less dilatation is usually found at the autopsy. Possibly some degree of incompetence has much to do with this. The auricular appendix is often blocked by firm thrombi, and the cavity of the auricle generally contains much A.M. and P.M. blood-clot.

Theoretically it might be considered that the left ventricle should show but little alteration, or even be diminished in size, but generally when examined post-mortem it presents some degree of dilatation, and not infrequently distinct hypertrophy.

MITRAL INCOMPETENCE.—In the normal heart the mitral valve effectually prevents any communication between the left ventricle and the left auricle during the systole of the former.

Two distinct forms of mitral incompetence, essentially different in etiology and mechanism, although presenting many clinical features in common, can be recognised:—

(i.) Incompetence from primary valvular disease.
(ii.) Incompetence from muscular failure.

Etiology.—According to the form of mitral incompetence, two chief groups of etiological agents are to be sought:—

1. That in which the curtains of the mitral valve or their tendinous cords are affected.

(i.) Infective Endocarditis.—The rapidly developed and often very extensive and destructive character of this malignant inflammatory process has already been indicated. The mitral segments, like the aortic, frequently suffer, and very extensive incompetence may be established, and that so speedily as often to give little opportunity for any effective compensatory changes.

(ii.) Simple acute endocarditis, generally of rheumatic origin, although often considered as giving rise to regurgitation, probably often allows of such rather from the associated muscle failure than the actual changes occurring in the valve segments—at least the muscle failure assists in the result.

(iii.) Chronic endocarditis, the consequence usually of rheumatism as already explained, not only leads to deformities, but is apt to produce loss of mobility, and a deterioration of the structures as an efficient mechanical valve. Althoug sclerosis of the mitral tends to produce incompetence, and that perhaps at a com paratively early stage in the cardiac history, yet in many instances the degree o compensation has been such that the case does not come under medical observatio until the mitral orifice has been considerably stenosed, and the then marke regurgitation is obviously occurring through an obstructed orifice. In all probability, however, some degree of regurgitation invariably occurs before the development of stenosis.

(iv.) Traumatic influences are said to be capable of establishing sufficient injury to the mitral valve as to produce incompetence, but it is difficult to obtain evidence in favour of such an assertion.

2. That in which the valve curtains are normal, but changes in the muscle substance lead to a widening of the mitral orifice. In advanced cases probably also some yielding of the structures forming the auriculo-ventricular ring may occur.

Mitral regurgitation from such causes may be of almost any degree of severity. In some the enlargement of the mitral orifice is slight and temporary, in others it is enormous and persistent. One group may be relatively curable; another incurable and incapable of lasting amelioration.

All conditions leading to a lowering of the nutrition of the cardiac muscle are to be considered instrumental in the production of this form of mitral insufficiency. Among such are the following: (i.) Febrile states; (ii.) Toxic conditions, especially alcoholism; (iii.) Anæmic affections; (iv.) Local inflammatory degenerative or other abnormal processes affecting the myocardium; (v.) Some chronic wasting diseases.

It may be well here to point out more explicitly that the toxæmic influence of many of the infectious diseases on the cardiac muscle may be such as to effect marked mitral insufficiency. Rheumatism, also, although attacking the endocardium in chief degree, can sometimes produce such myocardial enfeeblement as will lead to a considerable amount of dilatation, and amply to account for signs of mitral incompetence. The importance of muscular strain in such states as those above indicated, or indeed in any condition of cardiac debility, is self-evident.

Mitral incompetence due to gross lesion of the valve itself is, of course, strictly speaking, incurable, although adequate cardiac action, if compensation be established, may be maintained for years. Incompetence from muscle failure, on the other hand, may be to all intents and purposes curable, provided that the influences productive of the muscle failure can be removed and the state of normal nutrition re-established.

Morbid Anatomy.—As already indicated, two distinct classes are to be recognised:—(i.) Incompetence from primary valvular defect; (ii.) Incompetence from muscular weakness.

The valvular defects are such as have been previously described. They consist of a thickening, puckering, and shrinking of the curtains of the mitral, with usually more or less shortening, thickening, and induration of the tendinous cords. In a very considerable number of the cases which *clinically* presented the classic indications of mitral incompetence narrowing of the mitral orifice is found.

The muscle failure may manifest itself pathologically by dilatation of the ventricular cavity and simple enlargement of the auriculo-ventricular orifice, but these conditions will be best referred to later.

AFFECTIONS OF THE PULMONARY VALVES

Lesions limited to the pulmonary valves and orifice are rare. Sometimes, however, the pulmonary artery and valves are involved in association with or secondary to affections of the left side of the heart.

Two conditions are of clinical importance:—

(i.) Pulmonary stenosis; (ii.) Pulmonary incompetence.

PULMONARY STENOSIS.—Obstruction at the pulmonary orifice may be of (i.) congenital origin, or (ii.) acquired subsequent to birth.

Congenital pulmonary stenosis constitutes one of the commonest varieties of cardiac malformation. It is frequently associated with imperfection of the auricular and ventricular septa and patency of the ductus Botalli. All degrees may be met with up to complete occlusion of the orifice and atresia of the pulmonary artery.

It is often difficult, both clinically and pathologically, to distinguish between a stenosis of congenital or acquired origin.

Primary pulmonary stenosis, arising subsequent to birth, is exceedingly rare. When met with it is usually dependent on old endocarditis of rheumatic nature. In not a few of the recorded cases the etiology has been obscure.

The valves are thickened, often rigid, and even infiltrated with lime salts. The edges of the cusps may be fused and a funnel-shaped channel so produced, the apex directed upwards towards the pulmonary artery. The orifice may be diminished to a mere slit.. Vegetations may be present, and so still further narrow the orifice.

Occasionally in malignant endocarditis large fungating vegetations form on the pulmonary cusps and partially obstruct the orifice.

Obstruction has been met with at a point beneath the pulmonary valves. The walls of the conus arteriosus are thickened, rigid, and contracted, as from old ventricular endocarditis. There may be recent vegetations about the indurated region.

Obstruction has also been known to arise from chronic atheroma of the pulmonary artery.

In all cases of pulmonary stenosis the right ventricle will be found hypertrophied, and usually with its cavity dilated.

The pulmonary artery, as already indicated, while generally involved in the congenital cases of stenosis, does not usually present much change in the acquired form.

Pulmonary Incompetence.—Incompetence of the pulmonary valves may arise from the following causes:—

(i.) Primary disease of the valve segments. This may occur: (*a*) in pulmonary stenosis; (*b*) chronic endocarditis and sclerosed conditions of the sigmoid cusps unassociated with stenosis; (*c*) in malignant endocarditis; and (*d*) in the very doubtful cases of laceration from trauma.

(ii.) Secondary involvement of the valve: in (*a*) dilatation of the pulmonary artery; (*b*) pressure on the orifice and valves from without as very exceptionally in aortic aneurysm.

It will thus be seen that the mechanical defect of pulmonary leakage may arise from several causes. In some instances the condition is undoubtedly of congenital origin, but it is then usually associated with obstruction of the orifice.

Probably the so-called simple pulmonary endocarditis is of rheumatic origin. In not a few cases, however, of advanced aortic and mitral disease we have found a certain degree of induration of the pulmonary segments, and it seems quite feasible to believe that this thickening is due to excessive pressure, and is allied to atheromatous disease of the aorta.

Simple valvular disease of the pulmonary cusps presents the usual appearances of a chronic, inflammatory, and degenerative process. The segments are indurated, puckered, and do not approximate, but allow of free leakage.

The right ventricle and right auricle become hypertrophied and dilated. The pulmonary artery may be normal in size, dilated, or even contracted in some cases.

A relative or functional incompetency of the pulmonary valves may occur as a natural adaptation, as in cases of persistent high pulmonary pressure.

In advanced mitral stenosis, however, the pulmonary artery is more or less constantly distended, and dilatation occurring, may allow of regurgitation through the pulmonary orifice. In such cases, also, the pulmonary artery may be atheromatous.

Affections of the Tricuspid Valves

Primary or isolated lesions of the right auriculo-ventricular valves are exceedingly rare, while secondary impairment of their mechanical action is met with in the greater number of cardiac cases.

During fœtal development malformations and inflammatory processes are more liable to involve the tricuspid than the mitral valves, but after birth, although the tricuspid may suffer in connection with or secondary to the other valves, a lesion limited to them is quite exceptional.

The morbid conditions may be divided into (i.) Tricuspid stenosis and (ii.) Tricuspid incompetence. When the former exists the latter is also usually present.

TRICUSPID STENOSIS.—Tricuspid obstruction ranks amongst the rare forms of valvular lesion. The greater number of recorded cases have occurred in females. The age at death is usually between 20 and 30. It is often said to be of congenital origin, but of this we have grave doubts. In most instances it is associated with mitral stenosis. As a general rule the degree of stenosis of the tricuspid is much less than that of the mitral. In most of these cases the involvement would seem to be coincident and dependent on rheumatism. Tricuspid stenosis is also said to arise as a secondary lesion from inflammatory processes set up by back-pressure acting through the pulmonary circuit. It is probable that this is never the case.

The morbid anatomy of the stenosed tricuspid is almost identical with that of the similarly affected mitral, and hence does not call for any detailed description. The cusps are thickened, contracted, more or less adherent to one another, deformed, and sometimes connected into a funnel-shaped structure with induration and adhesion of the chordæ tendineæ, and the orifice thereby greatly narrowed. Vegetations may still further obstruct the orifice, and abundant deposition of lime salts may add to the rigidity of the sclerosed tissues.

The secondary results have already been sufficiently indicated, and need not be further dwelt on, except to point out that the greater part of the changes found in the chambers of the right heart are, generally speaking, due rather to the usually present and more important mitral stenosis than the tricuspid lesion.

TRICUSPID INCOMPETENCE.—Insufficiency of the tricuspid valves, allowing of regurgitation, is, as above indicated, the commonest of valvular defects. Indeed, in states of over-distension, such as may occur in the course of violent exertion, a leakage or "safety-valve" action may be considered as physiological.

For practical purposes, therefore, it is all-important to differentiate cases into two groups:—

(i.) Tricuspid regurgitation arising from muscle failure of the heart and independent of lesion in the structure of the valves.

(ii.) Tricuspid regurgitation dependent on morbid conditions of the valve segments.

Where the latter exists, the influence indicated in the former is only too apt to be sooner or later superadded.

Tricuspid regurgitation from relaxation or stretching of the fibrous ring constituting the base of the valve undoubtedly occurs, but usually has been for long preceded by asthenic or distinctly morbid affections of the muscular substance. Conditions of degeneration, inflammation, or like states impairing the contractile power of the cardiac walls and papillary muscles arise from a variety of agencies, as already indicated, and hence it is easy to understand how tricuspid incompetence may be consecutive to many different diseases and dissimilar lesions.

It is necessary also to insist on the paramount importance of influences acting on the muscle of the right ventricle through the pulmonary system. This arises either from local morbid conditions of the lungs, such as chronic bronchitis, emphysema, fibrosis, and the like, or from backworking of the effects of left-sided lesions and conditions of high arterial tension such as occurs in chronic Bright's disease.

But in a considerable number of the cases of tricuspid incompetence there is a distinct alteration in the valve segments. In one class the cusps will be found thickened, puckered, shrunken, and with induration of the chordæ tendineæ, and possibly calcareous deposition. Usually the mitral will also be involved to a greater extent, and frequently the aortic cusps. Here the valvular crippling is dependent, in the great majority of cases that are not congenital, on rheumatism or chorea.

Atheroma is sometimes said to involve the tricuspid valves, but we believe this to be quite exceptional.

In another class of cases the tricuspid valves become swollen, deformed, and more or less destroyed by a malignant endocarditis.

Tricuspid regurgitation being generally a secondary condition, the state of the heart will often be in great part dependent on the primary lesion, but in the cases in which the tricuspid incompetence is of primary origin, or at all events the most conspicuous feature, the right auricle will be found enormously dilated with a variable degree of hypertrophy of its walls, although sometimes they may be distinctly thinned. The venæ cavæ are usually much distended. The general effects have already been fully indicated.

III. Affections of the Myocardium

It is only of recent years that a true appreciation of the importance of lesions of the myocardium has aroused clinicians and pathologists to a more intimate study of the same. A clear recognition of anatomical arrangements and structural peculiarities constitutes an essential preliminary. Of special importance is the vascular supply. The structure and characters of the coronary vessels have, however, been described elsewhere and need not be referred to again, except to insist on their great importance in considering many of the most important pathological conditions of the myocardium.

In dealing with the processes of disease occurring in the heart frequent reference was made to involvement of the muscle substance. It will be well, therefore, in the present section to limit our consideration to the more important affections of the myocardium.

Cardiac debility, although a term which, pathologically speaking, is indefensible, nevertheless conveniently indicates a condition, congenital or acquired, of inadequacy without visible structural change. Asthenic states of the cardiac muscles arise often under the influence of deficient coronary arteries, and may even end in angina pectoris. Sometimes cardiac debility is temporary, but more frequently it is of long duration, and constitutes one of the forms of disability embraced under the comprehensive but indefinite clinical designation of "weak heart."

Atrophy and degenerations of the myocardium are very common. They are to be looked upon as evidences of either local or general malnutrition. In not a few instances the muscular degradation is dependent upon general blood states of anæmia or toxæmia. In other local conditions involvement of the coronary vessels is the determining cause.

Simple atrophy is said to occur as a part of the general wasting incident to old age, but certainly in healthy old people the very opposite seems frequently to be the case. Atrophy arises also in conditions of starvation and general malnutrition, and as a part of a more direct disease, such as fibrous transformation of the myocardium.

Special forms of atrophy have been described, such as:—

(i.) Fibrillary atrophy, where there is, as it were, an exaggeration of the longitudinal striation of the muscle fibres;

(ii.) Fragmentary atrophy or segmentation, in which a splitting up o the muscle cells is noticed, probably from a necrosis of the myosin; and,

(iii.) Pigmentary atrophy, where hæmatoidin granules of a brownis yellow colour appear about the nuclei of the muscle cells, and the myocardium becomes darker, harder, and lighter in weight.

Degenerations of varying degree are common, but have been alread sufficiently described.

Myocarditis or inflammation of the cardiac muscle is practically alway a secondary condition. It may develop in the course of infectious fevers o in association with septic and toxic states. It is also said to arise from a local extension, as from endocarditis or pericarditis.

Myocarditis is often described as being either (i.) parenchymatous or (ii.) interstitial; but such a distinction is somewhat artificial, as in most instances both muscle and inter-muscular substance are involved. The process may be (i.) general or (ii.) local. As regards intensity it is (i.) acute, (ii.) subacute, or (iii.) chronic.

Whatever may be urged on the ground of theoretical pathology, it is certainly difficult to draw any sharp distinction between certain forms of degeneration of the cardiac muscle cells and so-called parenchymatous myocarditis. Profound protoplasmic change in the muscle elements occurs in many toxæmic conditions, but particularly in typhoid, diphtheria, and septic fevers. It is important to remember that a like change occurs in acute rheumatism.

In some cases of "alcoholic heart" the muscle cells present characters indistinguishable from those met with in diphtheria. Influenza has of recent years amply proved itself, in many instances, a serious agent in affecting heart failure. The importance of pneumonia in bringing about rapid cardiac collapse, especially in muscle degraded by chronic alcoholism, is too well known to need more than a passing reference. But so-called parenchymatous myocarditis to a greater or less extent may occur in almost any disease of an infective character, or morbid condition associated with continuous pyrexia.

Special reference may here be made to the "alcoholic heart" so common among heavy beer-drinkers in male labourers, and such as draymen, market-porters, cabmen, and public-house employees, but from which spirit-drinkers are far from being exempt. Although the symptoms of cardiac failure may be of sudden or rapid development, more or less hypertrophy, and often very conspicuous increase in the muscle, is almost always present. The toxic influence of alcohol on muscle leads to degenerative changes, but the enormous quantities of fluid frequently imbibed must not be left out of consideration.

When a myocarditis proceeds to the formation of pus it is spoken of as a suppurative myocarditis, which may be localised, forming one or more abscesses, or more rarely diffuse. Such results from pyogenic infection. The micro-organisms may reach the myocardium by means of the blood, or by direct extension from the endo- or pericardium. Many are of the same nature as those met with in malignant endocarditis, and produce rapid degeneration and necrosis with a certain amount of inflammatory exudation.

Chronic Interstitial Myocarditis.—Under this heading it has become customary to include those conditions characterised by a formation of fibrous or indurated tissue, and when the true muscle elements have wasted or been replaced by sclerotic formation. But myocardial sclerosis is by no means always the result of a true inflammatory process. Hence the above term is hardly sufficiently inclusive to be absolutely accurate.

Arterio-sclerosis of the coronary arteries must be credited with the greater number of cases of fibroid heart. The influence of gout, lead, syphilis, Bright's disease, alcoholism, over-work and old age, in directly leading to such is discussed elsewhere, and calls for no further consideration here.

The myocardial sclerosis may be (i.) local or (ii.) diffuse. When local, and under the influence of arterial supply, the left ventricle usually suffers to the greatest extent. The indurated areas appear as firm, pale, fibrous patches which microscopically present the characters of dense cicatricial-like tissue.

Cardiac Aneurysm.—Local bulging of the myocardium is usually dependent on antecedent disease of the muscle ending in fibroid induration. Exceptionally, however, a cardiac aneurysm forms before fibrous transformation has occurred. The endocardium is also sometimes found bulging between the muscular fasciculi. Cardiac aneurysms are most frequently situated in the walls of the left ventricle, on the anterior aspect and near the apex; but they also arise on the posterior wall and even in the septum, when they may bulge towards or into the right ventricle. In size they vary from that of a marble to a hen's egg, and occasionally may even be of larger dimensions.

Aneurysms in connection with the valves and local bulgings on the coronary arteries are referred to elsewhere.

Hypertrophy and dilatation may occasionally exist separately, but usually occur in combination, constituting the commonest cause of cardiac enlargement. Hypertrophy may always be considered a secondary condition, and is in most instances to be looked upon as compensatory in nature. Dilatation is generally secondary, and mainly indicative of myocardial inadequacy.

Hypertrophy may arise from (i.) intrinsic or intra-cardiac conditions, such as valvular affections, and perhaps sometimes "adherent pericardium," and (ii.) extrinsic or extra-cardiac conditions, such as arterio-sclerosis and chronic Bright's disease. In hypertrophy, consequent on toxic influences, as from alcoholism and excessive smoking, and long-continued neurotic palpitation, as for instance in Graves' disease, probably dilatation is to be considered the first and essential lesion, hypertrophy occurring rather as a later effect. The hypertrophy met with in those employed in laborious occupations, and that occurring in pregnancy, is to be looked upon as in the borderland of the physiological. Hypertrophy is generally to be construed as a reparative or compensatory effort, while dilatation works in the direction of a break in the restorative mechanism except in the case of aortic incompetence, where to a certain degree it is advantageous.

Formerly three varieties of hypertrophy were described—(i.) *simple*, where although the myocardium was increased the cavity remained of normal dimensions; (ii.) *eccentric*, with both walls and cavity increased, the form now better described as "hypertrophy with dilatation"; and (iii.) *concentric*, where the cavity was thought to be diminished, but which is now recognised as due to post-mortem contraction.

The whole heart may be hypertrophied, when it is aptly described as "bovine"; if the right ventricle is chiefly involved a "quadrate" form will be assumed; and when the increase in muscular development predominates in the left ventricle a "conical" heart results, provided, of course, the left ventricle has not become rounded and lost its apex.

It is impossible here to describe in detail what may be termed the local distribution of cardiac hypertrophy. Brief reference can only be made to some few points. The walls of the left ventricle manifest the greatest muscular development, particularly in disease of the aortic valve. In aortic stenosis hypertrophy predominates and may exist alone for a long period, but in incompetence it is associated from the first with dilatation, which, indeed, in a sense may be regarded as compensatory, though hypertrophy must speedily follow. In aneurysm of the thoracic aorta no hypertrophy may occur, provided the aortic valves remain competent. In "granular" kidney enormous and almost pure hypertrophy arises, but even here the right ventricle participates to some extent. Hypertrophy of the left auricle occurs in mitral stenosis. Obstructive diseases of the pulmonary circulation lead to great hypertrophy of the right ventricle, but diseases of the aortic and mitral valves, especially mitral stenosis, are a common cause. In congenital lesions very extensive hypertrophy is often apparent. It may be desirable here to add that even in "right-sided" cases of hypertrophy more or less implication of the left ventricle will also be found, as indeed would be expected when the "solidarity" of the heart is considered.

A hypertrophied heart may assume considerable proportions and increase greatly in weight. The 9 ounces of the normal heart may frequently be doubled, often reaching 25 or 30 ounces, and have been known to touch 46½ ounces. Much discussion has taken place as to the actual change in the muscle elements. It would appear that there is an increase both in the size and number of the cells. *Dilatation* is occasionally detected as a primary condition, but usually occurs in combination with more or less hypertrophy, the latter process tending to stay the former. Sometimes dilatation may occur as an acute condition, as for example in the kidney affections of scarlet fever, but usually it is rather of a chronic and progressive development. While the predisposing causes are many and often obscure, the exciting influences may be readily grouped into two classes: those acting (i.) by diminishing the contractile power of the muscle, and (ii.) by increasing the pressure to be overcome. In the former class may be placed conditions of atrophy and degeneration, some forms of infiltrations, and most inflammatory states of the myocardium. Certain cavities may be more markedly dilated than others, but frequently the whole organ is involved by a general dilatation. When only a part of a chamber is affected it is usual to speak of it as an "aneurysm of the heart."

SYMPTOMATOLOGY

The symptoms of heart disease, whatsoever be its form, are very similar, and seeing that they for the most part depend on the same kind of disturbance in the circulation (resulting in venous stasis) of the different organs of the body, it is not surprising that this should be the case. It is on physical examination mainly that we depend, as already stated, for the differentiation of the causes of such disturbance that lie in the heart, but not entirely on this mode of investigation, seeing that it alone will sometimes entirely fail to enable us to form a diagnosis worthy of the name, which attention to the history of the patient will enable us at once to accomplish. This statement will be made clearer by taking two actual cases in illustration:—A man aged forty complains of the following symptoms: shortness of breath on any exertion, dropsy of the lower extremities, and some uneasiness referred to the right hypochondriac region, the first progressive and of six months' duration, the second and third of six weeks' duration. *Signs:* On physical examination the veins of the neck are seen to pulsate while the patient is upright: the heart is found to be somewhat enlarged; over its apex there is a systolic murmur following the first sound, which latter is alone audible to the inferior angle of scapula behind, and the pulse is irregular. Now these conditions, we shall find, are common to two very different forms of heart disease—mitral stenosis and muscle failure. But the *history* of the patient will enable us at once to decide between them, for at the age of fifteen the patient had a severe attack of acute rheumatism and has been a temperate and careful liver. In short, the *symptoms* render cardiac disease probable; the *signs* confirm this opinion, but it is the *history* that enables us to make the diagnosis of mitral stenosis by informing us of the old attack of acute rheumatism which renders that lesion probable under the circumstances. Again, in a man a few years older the symptoms and signs are closely similar, but the history excludes rheumatism and chorea, and proves that his "wind" was good up till the time of his leaving the army six years before, in which he could perform arduous drill with ease to the end, and reveals the fact that since leaving the army he has been a confirmed beer-drinker, and as a consequence has neglected his proper food. The case is one of alcoholic muscle failure of the heart.

But let us suppose that the patient is sixty years of age and had an attack of acute rheumatism at thirty-five, while his cardiac symptoms are of comparatively recent date, say five years' duration, the same physical signs will in the presence of the history leave us somewhat in doubt as to the possibility of there being stenosis as well as senile muscle failure. Still the age at the time of the rheumatism and the recent development of the symptoms of disturbance of the circulation render it probable that the case is one of primary muscle failure, and that the patient escaped any serious consequence of endocarditis. If the patient had aortic incompetence as his lesion, a similar question of pathological diagnosis may arise, and again probability would be in favour of the origin of the disease being degenerative and arising in the vessel, rather than rheumatic and arising in the valves.

As regards symptoms, then, practically all forms of heart disease may be studied together: it is the physical signs they present that accomplish their separation in the light of the past history of the patient and a knowledge of pathology.

There are three symptoms that stand out pre-eminently from among the many to be considered: these are *dyspnœa*, *dropsy*, and *enlarged tender liver*, and they may be regarded as the cardinal symptoms of heart disease.

1. Of the three, *dyspnœa* is probably always the first to manifest itself, though it occasionally happens that patients take no heed of it until dropsy supervenes, which immediately alarms them and renders them observant. In such cases careful inquiry will almost invariably elicit evidence of failure of "wind" on exertion for some time before the appearance of dropsy. So frequently is this the case that one may be pardoned for doubting the statement of the few patients who obstinately deny having had any dyspnœa before they noticed dropsy. Many people seem to have great difficulty in defining their sensations, and yet give a description of sensations that points strongly to there having been among them the feeling that most people would describe as "shortness of breath" or "breathlessness," without having recognised it as "air-hunger," to use the expressive term of the Germans. It is on *exertion* that the first experience of dyspnœa usually occurs. We shall have, however, to describe certain distressing forms that occur independently of any exertion, but it may be taken for granted that the "wind" of those who suffer from them is always at fault on exertion. *There is no indication of the well-being of a heart to be compared in importance with "goodness of wind."* If the physician is *assured* of this he need hardly examine the heart, for "sound wind" and "an unsound heart" may be regarded as incompatible conditions. Ascent, either of steps or a simple incline, throws a peculiar strain on the heart apparently, and discovers the crippled heart with unfailing certainty. It must be remembered, however, that a man who has followed a sedentary occupation and habitually avoided active exercise, may have "bad wind" without any actual disease of the heart, and simply as the result of his having persistently given the organ the least possible amount of work to do, but such a heart is capable of great improvement under treatment by processes analogous to that employed in athletic training.

A man may have a cardiac lesion—say mitral stenosis or aortic incompetence—and yet suffer no inconvenience in consequence, so long as he is subjected to no strain; but he will not ascend a flight of stairs without some degree of dyspnœa over and above that experienced by the average healthy individual; the effort betrays his weakness, which without such exertion may be entirely latent, thanks to the vigour of the heart muscle.

Breathlessness on slight exertion, then, is the first indication of cardiac failure in general, but in certain cases this is not all, and the patient while at rest in bed during the night is seized—often awakened by—a distressing paroxysm of breathlessness which compels him to sit up and "struggle for breath." This type of dyspnœa is especially apt to occur in cases of Bright's disease and gout—in fact, in cases of muscle failure of the heart in combination with a fairly high degree of arterial tension, and the writer is disposed to believe that associated with such attacks is an abrupt rise of the arterial blood-pressure whereby the heart is seriously embarrassed.

Another form of dyspnœa often noticed in similar cases is that in which the stimulus of volition seems necessary to maintain the activity of the respiratory centre, for when the patient falls off to sleep his respiration ceases, and he becomes cyanotic, often showing in consequence slight convulsive movements of the hands. These are soon arrested on his awaking, gasping for breath. Another remarkable type of dyspnœa that comes on during repose is known as Cheyne-Stokes respiration, in which pauses in respiration occur at definite intervals, each one being followed by the resumption of respiration at first with the shallowest of inspirations, which become gradually both deeper and more frequent until the acme is reached, when the inspirations become less deep as well as less frequent, until another pause sets in, to be followed in turn by another series of respirations of ascending and descending type, and so on. This type of dyspnœa is specially common in the heart failure of chronic Bright's disease (granular kidney). It has also been supposed to be associated with fatty heart. During the paroxysm of dyspnœa patients will sometimes spring out of bed at the maximum of their distress. On the other hand, respiration of "ascending and descending" type may occur without any subjective distress of any kind. A rare and often overlooked form of cardiac dyspnœa, if such it can be called, consists of an occasional very deep inspiration or sigh. Long ago this peculiar sighing respiration was recorded as a sign of "fatty heart." Certainly it belongs to the more obscure cases of cardiac failure, which may terminate by sudden fatal syncope, or which may be revealed by the ordinary symptoms of venous stasis only shortly before the end. The writer cannot say if there has been any stoppage or alteration of respiration immediately preceding these deep inspirations, which occur after long intervals and apparently in the midst of quiet respiration.

2. *Dropsy.*—The second cardinal symptom of heart failure, dropsy, is probably invariably preceded for a longer or shorter period by dyspnœa. It is likely, however, that the tissues through which and into which the exudation of serum takes place undergo some nutritive change, which favours the process. This "tissue-element" in the production of dropsy seems to vary in activity, its working being less apparent in cases of chronic valve lesion, as the rheumatic, and most apparent in the dropsy associated with the muscle failure that results from alcoholism. The tendency to tissue degeneration and vaso-motor palsy promoted by this habit is notorious. In the simple rheumatic case the dropsy begins where we naturally expect there being the greatest venous stasis—the lower extremities—and spreads upwards as the conditions that occasion it become intensified. But the dropsy of the alcoholic heart-case is characterised by extraordinary caprice both in its first and later localisation, and ultimately by its as extraordinary extent of distribution. The first peculiarity is exemplified by such rarities as the scrotum being for a time alone affected, the second by the whole surface of the trunk becoming very dropsical, so that a deep and long-lasting indentation can be made with the finger all over; sometimes the scalp is similarly invaded. In a case of the kind the œdema is sometimes particularly noticeable over the sternum, where a massive pad, pitting an inch or more deep on pressure, is often formed. Over the lower back is a common localisation for dropsy, and this "pitting" over the sacrum may be found actually when there is no dropsy of the lower extremities. This, of course, happens usually in patients who have been recumbent for some time. The frequency of kidney complication towards the end of cardiac cases is very great, and the writer at one time placed this question before himself, Do the upper extremities ever become œdematous in heart cases in which there is no albumin in the urine? and he was soon able to answer the question in the affirmative, though the occurrence is rare. Even dropsy of the face may occur under the same circumstances, though unquestionably it is very much

less common than in Bright's disease of the kidneys; in the former case it is never an early dropsy, and is probably always associated with an extensive development of dropsy elsewhere. A comparison of the two dropsies, that dependent on kidney disease and that dependent on heart disease, results in bringing into relief rather their points of resemblance than their points of difference; we exclude from consideration the numerous cases of heart failure secondary to Bright's disease, cases into which a cardiac element is introduced.

Serous exudation into the subcutaneous cellular tissue is very likely to be accompanied by serous effusion into one or more of the serous sacs—pleuræ, pericardium, and peritoneum—but it is often difficult to be certain that a given effusion is a dropsy in the ordinary sense, and not of inflammatory origin. This difficulty is specially met with in the case of pleural effusions, the lung complications in heart disease being so numerous and so apt to set up secondary pleuritic effusion while apparently primary pleuritis is far from rare. Hydrothorax in the sense of simple pleural dropsy is generally bilateral, but so may pleuritic effusion be, while simple dropsy may occur on one side only, because the pleural sac on the other side has been obliterated by old adhesions, in which case the lower part of the corresponding lung becomes œdematous. It is a curious fact that ascites is apt not only to be specially pronounced in cases of mitral stenosis, but may actually be the only dropsy present. The circumstance is no doubt associated with the special proneness of the liver to suffer from venous stasis in that disease, and with the long duration of the lesion permitting secondary changes to be developed in the organ.

3. *Enlargement of the Liver.*—This is so marked a feature of the disturbance of the circulation resulting from heart failure that it is fully entitled to be placed with the other two symptoms we have considered as cardinal. Stokes admirably described the condition in 1854.[1] Speaking of an exacerbation in a heart case, he says: "It is under these circumstances that the already enlarged liver exhibits a rapid increase of tumefaction, in a few hours descending far into the abdomen, yet on the subsidence of the attack returning to its ordinary volume, when it may be felt as a flat indolent tumour extending for an inch or more below the false ribs" (p. 257).

When we consider the peculiarities of the portal circulation it is not surprising that the liver should be specially susceptible to the venous stasis resulting from heart failure. That the swelling of the organ, noticeable under the circumstances, is due essentially to the vascular engorgement, is plain not only from the rapid diminution of the organ that can be traced at the bedside when the general circulation is restored, but also from the result of post-mortem examination of patients, who die with the organ engorged, but whose livers collapse on cessation of the circulation. The late Dr. Murchison divided the enlargements of the liver into the *painful* and *painless* varieties, and the enlarged liver of heart disease certainly belongs to the painful variety, but the pain is slight till the organ is pressed upon. Otherwise the organ is smooth, but feels somewhat indurated. This apparent induration (as the tenderness probably) is no doubt accounted for by the tightening of the capsule of the liver as its contents are increased under pressure, for it rapidly diminishes, and often disappears in a few days—in fact as soon as the circulation is restored and the venous stasis relieved. We have incidentally referred to ascites being occasionally the only dropsy present in cases of mitral stenosis,—generally cases of long standing,—and the fact is undoubtedly associated with another fact often observed in cases of the kind, namely, that the liver may be found greatly enlarged and tender on pressure while yet there is not—nor has been—a trace of dropsy of the lower extremities. There can be little doubt that the liver and portal circulation suffer specially in the venous stasis of mitral stenosis, though why this should be so is not clear. *Icterus* is exceedingly common in the later stages of cardiac disease in association with venous engorgement of the liver. The stools, however, usually contain bile. When there is marked cyanosis, as well as jaundice, the patient's countenance is apt to assume a greenish tinge, that to the experienced observer is peculiarly characteristic of the final stage of cardiac disease.

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[1] *The Diseases of the Heart and the Aorta.* Dublin, 1854.

Palpitation, or the too forcible or too frequent beating of the heart, whereby its action becomes perceptible to the individual, is an unimportant symptom in the majority of cases of organic heart disease, although it occupies a high position in the estimation of the laity. Moreover, in some cases of organic disease it is a cause of much distress. The writer has seen this result most frequently in cases of aortic incompetence, in which, notwithstanding huge enlargement of the left ventricle, the right side of the heart was little involved. Also in Graves' disease, which often leads ultimately to cardiac dilatation too, he has witnessed the symptom in most distressing severity. Patients often confuse various disturbances of their cardiac action under the term palpitation, which agree only in one respect, namely, that they make the individual aware of the movement of his heart. Thus the too forcible beat that follows an intermission or an abortive beat, and the peculiar "fluttering" sensation that often accompanies irregular action, are frequently described as "palpitation," so that the physician has to be careful not to accept the term in its proper significance too readily. Sometimes the patient seems to be hyperæsthetic, so that he is aware of his heart's action, though to the observer it seems neither unduly forcible nor sufficiently frequent to occasion the sensation, while it is quite regular.

Cardiac Pain.—The great pain associated with heart disease is angina pectoris, which seems unquestionably dependent—absolutely as far as the writer's own experience goes—on obstruction either at the orifice or in the course of the coronary arteries, one or both. Angina pectoris, however, is specially described in a separate article in this work. (See "Angina Pectoris," vol. i.) But it is astonishing how frequently the subjects of morbus cordis complain of local pain in the region of the affected organ. The writer is aware that the heart itself is stated by physiologists to be directly insensitive to pain, but he has good grounds for questioning the statement in disease. It may be that though in health the heart is insensitive, it acquires sensitiveness to pain in disease. That the heart *itself* does in disease become painful, though not severely, the writer cannot doubt in view of his clinical experience. Such local pain as is complained of by genuine heart sufferers is certainly seldom or never acute and severe, while it is often accompanied by some deep local tenderness over the apex-beat as well as by superficial tenderness to pressure over the second and third left costal cartilages, although the latter is not properly direct tenderness, but is associated with the relationship existing between the visceral and peripheral nerve-distribution proceeding from the spinal segment concerned in cardiac innervation, which is situated in the upper dorsal region.

Pulmonary Conditions.—In correspondence with the early dyspnœa experienced by sufferers from cardiac disease the consequences of lung congestion soon manifest themselves. Among the earliest of these is a hitherto unwonted tendency to bronchial catarrh, with its ordinary symptoms and physical signs: the latter rhonchi and, in severe attacks, moist sounds, which are apt to be most abundant at or to be limited to the bases of the lungs. As such attacks succeed one another these moist sounds linger longer after each attack, until their presence may become habitual, while the rhonchi from time to time clear away. In other cases, with neither present evidence of nor distinct history of bronchitis, moist sounds are found at the bases of the lungs, the bubble they denote being evidently of comparatively small size, while in still other cases a very fine bubbling sound limited to the end of inspiration, to which the term *crepitation* or "vesicular bubble" (Skoda) can hardly be denied, is persistently present for long periods, while rise of temperature, indicative of inflammatory action, is entirely absent. The explanation of these sounds, which are usually heard equally on both sides, is no doubt an œdematous condition of the lung-bases. Nowhere, so well as in the lungs, do we see illustrated the pathological law that venously congested organs are prone to become inflamed.

A large number of sufferers from chronic cardiac disease are carried off in the end by a pneumonic complication, and such processes are often associated with "pulmonary apoplexy," which will be considered in another part of this article (under Embolism).

There is little or nothing distinctive in the sputa of the cardiac sufferer from bronchitis; they present the ordinary characters of "bronchitic" sputa.

Little less important than the lung conditions are affections of the pleura in chronic heart disease. Effusion is usually the most important fact, and such effusion may be of inflammatory origin, or a simple dropsy as that of so many other parts. Pleuritis without effusion is usually secondary to some intra-pulmonary pathological state, such as embolism or pneumonia, when it is of quite subordinate importance. The possibility of one or both pleural sacs having become obliterated as the result of adhesions from old pleuritis must be borne in mind. When such obliteration has taken place, instead of the usual hydrothorax, œdema of the lung occurs. As regards the physical signs of hydrothorax, it is perhaps unnecessary to point out that the fluid in the case of a simple dropsy of the pleura is just as immobile as that of a pleuritis, when the patient's posture is changed, the old view, that adhesion of the pleural surfaces above the fluid, in the latter case, was the cause of the immobility, being quite untenable. In both cases the dull area corresponding to the fluid presents a convex boundary upwards. Vocal fremitus is always diminished, but by no means always lost. The naturally stronger fremitus on the right side must be remembered. As regards auscultation over the dull area there may be diminution of breath-sounds in all degrees up to silence, or there may be exquisite, though usually not loud, bronchial breath-sound and bronchophony or ægophony. The presence of bronchial breath-sound may be taken to mean that a considerable portion of lung has been rendered airless. It is important to remember, in a case in which there is question of tapping, that a few moist sounds or even friction, when the fluid is of inflammatory origin, must not deter us from exploring, if otherwise the evidence of the presence of fluid is strong. Such adventitious sounds are probably only met with when the effusion is partial, the sounds being produced in the still air-containing lung above the effusion in the one case, and likewise above the effusion in the other case, at a place where the inflamed pleural surfaces are in contact, the friction sound produced between which is readily conducted along the chest wall. The writer has drawn off fluid from a perfectly dull area, over which almost loud friction sound was audible. The accumulation of fluid in the pleura is always an important fact in a heart case, for a moderate effusion may seriously interfere with the already embarrassed pulmonary circulation, and the removal of even such an amount of fluid may afford great relief. On the other hand, just such a moderate effusion of considerable standing may almost suddenly increase, so as to render the whole lung airless. How often is the physician appalled at the post-mortem of a chronic heart case, by finding one pleura full of fluid, the corresponding lung airless, and the other lung œdematous, when he had perhaps only thirty-six hours previously found the fluid in the pleura to reach less than half-way up the chest, and the opposite lung to be free from any serious amount of œdema! This insidious though rapid development of effusion is very often the final pathological event in long-standing cases of chronic heart disease, and it is one of the accidents against which the physician should ever be on guard.

The *hæmoptysis* of heart disease finds its appropriate place of description among the symptoms of the embolic process. That hæmoptysis does occur, however, without the accident of embolism, and apparently as a result of the general congestion of the pulmonary circulation only, is almost certain. Of this origin it is seen specially in the young subjects of mitral stenosis, in whom it is not rarely the first symptom that brings the patient under medical supervision.

Kidneys.—With the exception, perhaps, of the lung, in no organ are the changes that result from venous stasis of greater practical importance than in the kidney. In the great majority of cases of heart disease there is *albuminuria*, and in the majority of cases with albuminuria the pathologist would report that the kidneys were only "venously congested"; in a certain number he would find evidence of long-standing chronic disease of the kidney (granular kidney most commonly), while in the rest he would pronounce the organs to have become recently inflamed, and to present the macroscopical appearances of a tubular or "mixed" nephritis. Clinically, the difficulty of distinguishing between these pathological states is often very great indeed. The writer remembers an old man, suffering from general anasarca, engorged liver, and pulmonary apoplexy, the results of muscle failure of his heart, whose urine was high-coloured, deposited deep-coloured urates, and had a specific gravity well over 1020, and yet contained a mere trace of albumin. Surely under the circumstances a very much larger quantity of albumin would have been no necessary indication of kidney disease? Yet this patient had exquisitely granular and contracted kidneys. The abundance of albumin, on the other hand, that is quite commonly present towards the end of heart cases, without there being any macroscopical nephritis, is notorious. The mere presence of casts will not decide the question whether we have to do with a "congested" or an inflammatory or a degenerate kidney, although abundance of casts, especially other than hyaline, points strongly to there being more than mere congestion of the kidney. As to the nature of the casts, apart from their number, hyaline and slightly granular casts are most common in cases of the simply congested kidney, while many epithelial and leucocyte casts point decidedly to actual nephritis. In long-standing cases of granular disease of the kidney with secondary muscle failure of the heart, it is, however, often extremely difficult to find casts in the urine at all, because there are so few present. Thus to determine the presence of actual kidney disease in cases of heart failure, in one form or another, may be a practically impossible task. Occasionally the establishment of the diagnosis of kidney disease is at once attained, not by any examination of the urine, but by the use of the ophthalmoscope, "albuminuric retinitis" being so revealed. The urine of pure heart cases is generally high-coloured—apart from the presence of bile—the character no doubt proceeding from the congested state of the liver as well as being promoted by the concentration of the secretion. A persistently pale and comparatively scanty urine is, again, suggestive of advanced kidney disease, though under the circumstances it is not likely that cardiac failure will be pronounced. The writer is not aware that those who have devoted special attention to urinary casts can assert from the kind of casts present in a heart case that the limit of mere "venous congestion" has been passed and that true nephritis is established. No doubt abundance of casts, and specially of casts other than hyaline, which have least value, points strongly to the passage having been made, or to there having been *old* nephritis. The frequent occurrence of embolism of the kidney in heart disease must be borne in mind with regard to albuminuria, and especially hæmaturia. In one form of heart disease hæmaturia is specially frequent—not a transient appearance of blood, as in embolism, but one usually persistent till the end of the case—in malignant endocarditis.

The *spleen* suffers little from venous stasis in cardiac disease, evidently because of the ready distensibility of the liver, which is interposed between it and the obstruction. Clinically it seldom happens that the spleen is found considerably enlarged from this cause, and when it is the liver has probably undergone some secondary change, impeding the circulation *in situ*. When the primary obstruction is situated in the liver, as in interstitial hepatitis, on the other hand, enlargement of the spleen is comparatively common. When the spleen is found markedly enlarged in a heart case the occurrence is so unusual that special circumstances are at once suggested: either there has been recent large infarction of the organ, or there is septic endocarditis, which is associated with enlargement of the spleen in the same way as enteric fever is similarly associated, and the enlargement of the spleen is thus not the result of mechanical interference with its circulation—of venous stasis. Whenever the enlargement is considerable the lower end of the organ can be easily felt below the costal arch, while its descent downwards and towards the right, with inspiration, is easily ascertained.

Stomach Symptoms.—The stomach in heart-failure being specially subjected, like the other organs in the distribution of the portal circulation, to venous stasis, becomes prone to suffer from catarrhal inflammation of its mucous membrane. Attacks of catarrh vary in their acuteness, and in some cases a subacute or chronic condition is assumed, generally the result of faulty treatment, and specially in the matter of feeding. It often happens that a patient with advanced heart disease has been by habit a good feeder, and may remain so even up to the end. The writer well remembers how the fact struck him, coming to a general hospital after two years' work in a fever hospital, the association of intense illness and the ability to eat and enjoy a meal being of course utterly unknown in fever practice. When there is no gastric catarrh it is not a rare event to see a patient, in the last stage of heart disease and not far from being *in extremis*, eating apparently with relish an ordinary meal. But even in such a case there have usually been attacks of sickness and vomiting during which there has been positive loathing of food. In these attacks the tongue usually becomes thickly coated, and there is often a peculiar odour in the breath—suggestive of that of chloroform—while the urine will often show a port-wine colour on the addition of the liq. of the perchloride of iron. This coloration is apt to be interfered with by the precipitation of phosphate of iron, which dissolves on further acidulation. This "acetone" odour in the breath—sometimes in the urine—of a patient with heart disease leads the expert observer at once to seek for other evidences of gastric catarrh. It is possible that such attacks are often immediately set up by inappropriate and irritating food. In a case of the writer's—an advanced one of mitral stenosis—the patient, a girl, had surreptitiously obtained an orange from her neighbour, and seeing the nurse approaching, gulped down the greater part of it in a mass, which was found post-mortem in her stomach seventy-two hours later, the symptoms of a most acute gastric catarrh having supervened, and speedily brought about a fatal termination. Even in this place the writer would call attention not only to the uselessness, but the actual harmfulness, of continuing the attempt to feed the patient by the mouth during severe gastric catarrh accompanied by vomiting. On the other hand, if the stomach is subjected to no medicinal treatment, but is simply allowed to rest absolutely—what feeding is necessary being accomplished per rectum—recovery is usually speedy.

Among the clinical symptoms of angina pectoris the eructation of flatus towards the end of the attack has long been noticed, and there seems to be a curious association between eructation of "wind" and unmistakable cardiac suffering. So much is this the case, that in questioning uneducated patients, the subjects of heart disease, with regard to their "wind" in the athletic sense of the word, they often misapprehend the application of the term, and enter into a lengthy description of their suffering from abdominal distension and the relief obtained when they can belch up the peccant "wind." The frequent association of the two conditions—heart disease in some form and abdominal distension with "wind"—at least the feeling of it—is too common to be ignored, whatever be its explanation. The subject is one of considerable interest with regard to diet in heart disease.

The intestines are no doubt apt to suffer from catarrhal conditions in a manner similar to the stomach, but it has become so much a custom to give at least occasional purgatives in the treatment of heart disease, that the influence of these in the production of intestinal catarrh has to be kept in mind. Apart from the use of purgatives, constipation is, perhaps, a more commonly troublesome symptom than looseness of the bowels. It often happens that, while there is habitual constipation, diarrhœa sets in from time to time, probably the result of irritating substances being formed in the intestinal tract. Here, as in the case of the stomach, the importance of the manner of alimentation of the cardiac sufferer is immense.

Hæmorrhage from the Portal System.—In severe gastric catarrh a little blood may appear in the vomited matters, but such hæmorrhage is usually very scanty. It occasionally happens, however, that a profuse bleeding occurs in a heart case per rectum, and is preceded by pain and a tendency towards collapse. In the worst of such cases embolism or thrombosis of large mesenteric arteries is the likely lesion to be found post-mortem, but more than once the writer has known profuse hæmorrhage from the bowels to occur in heart disease without there being found post-mortem any lesion to account for it.

Cerebral Symptoms.—Towards the end of cardiac cases mental disturbance is not uncommon, and assumes different forms, no doubt determined by the hereditary and personal predisposition of the individual. The writer doubts if any special form of mental derangement can be definitely ascribed to cardiac disease, much less to special forms of it. Painful delusions of suspicion are common, and under their influence patients will rise from their beds and subject themselves to efforts they might be thought incapable of. It happens as a consequence that a patient by thus throwing a strain upon his enfeebled heart may render impossible further rally, though the case is often nearing its end before the supervention of mental derangement. The many forms of heart disease, and the multifarious associations of the disease, must introduce toxic and other conditions as capable as the disturbed circulation of disordering the functions of the brain. For instance, the extreme cyanosis accompanying the cardiac failure of pulmonary emphysema is often associated with bad dreams, in which the patient vociferates loudly, so that in a ward he becomes an unmanageable nuisance to his neighbours. In the heart failure of Bright's disease, again, toxic conditions, determined by the kidney disease, probably have much to do with the mental disturbance, which resembles rather that of the "typhoid state" in general, than that associated with venous stasis. Active delirium, again, is a common accompaniment of the hyperpyrexia of acute rheumatism, so apt to be associated with endo- and pericarditis. Perhaps the most definite association, as cause and effect, of heart disease and cerebral disturbance is the hemiplegia resulting from cerebral embolism, which will be considered later.

Epileptoid and syncopal seizures are common as the result of sluggish cerebral circulation in cases of bradycardia. In extreme aortic incompetence mental disturbance is perhaps specially common, and in its production the wide variation between the maximum and minimum blood-pressure may play a part. The cyanosed, bloated face of the sufferers from extreme mitral stenosis or the cardiac failure of pulmonary emphysema contrasts strongly with the pale face of the subject of free aortic incompetence, and it seems reasonable to suppose that the two physiognomies reflect to some extent the mode of interference with the cerebral circulation, notwithstanding its peculiarities.

Active mental derangement is too often a lethal indication in cardiac cases, and the fact is to be explained by this derangement being a late symptom, and therefore likely to be associated with the more lethal conditions of other important organs, including the heart itself, and specially by the very grave opposition to the treatment of the case it offers. Thus if a patient in the late stages of cardiac disease insists upon going down and up stairs on his own legs, the effort may prove speedily fatal to the already over-taxed heart, and the medical attendant has no choice except between heavily narcotising him (not always a safe and easy thing to do) and restraining him by the hands of attendants. Strapping any patient down in bed is a relic of the barbarous ages that have hardly quite gone, but to strap down a cardiac case would simply mean manslaughter, so certainly and speedily would it determine hypostatic congestion of the lungs. After a prolonged forced sleep it often happens that the patient wakes up, if not *compos mentis*, at least amenable to the care of his attendants, but occasionally the morbid activities themselves awake likewise as if refreshed by rest.

Thrombosis of large veins is an occasional result of cardiac disease producing in the lower extremities the conditions so well known as "Phlegmasia alba dolens" (though it is not always painful), liable to occur after parturition, fevers, etc. In heart disease the accident is to be regarded as of very unfavourable prognosis, not so much because of its intrinsic gravity as of the state of general circulation and vitality it denotes. Moreover, the possible detachment of particles of clot and consequent pulmonary embolism has to be borne in mind. The dropsy that results from it is distinguished from cardiac dropsy by its being usually limited to one extremity, or if involving both extremities, by its being greater on one side than the other. Cardiac dropsy is, however, not invariably symmetrical, and the cause of such asymmetry is not always clear. It is obvious, however, that in a cardiac case ordinary dropsy is likely to be present, which the dropsy of the thrombosis only complicates. The patient's complaining of pain at the seat of thrombosis will often lead to the discovery of the accident, and when a cardiac sufferer complains of pain in a limb it is always necessary that the vessels—both veins and arteries—should receive careful physical examination for vascular obstruction. A more or less elongated mass or cord may easily be felt in some cases, while in others the seat of the obstruction has to be inferred from local tenderness and the disposition of the dropsy.

THE EMBOLIC PROCESS

The plugging of peripheral vessels in the various organs by particles, usually of fibrin, from the cardiac cavities or from diseased surfaces of valves, plays an exceedingly important part in the symptomatology of cardiac disease. The accident, as it may be called, of embolism probably always implies either stasis of blood in one or other of the cavities of the heart, as a result of which, fibrin is apt to be deposited from the blood in some of the recesses of the cardiac wall, or the exposure of some abnormal and devitalised endocardial surface, usually of the valves, to the blood-current, whereby fibrin is deposited, as happens in the case of rheumatic and septic endocarditis. *Cerebral embolism* is a common accident in mitral stenosis, and the source of the plug is usually a tiny clot detached from a recess in the wall of the left auricle. But cerebral embolism may occur also in cases of simple dilatation of the heart, the source being either the left ventricle or auricle, in either case presumably in a state of "systole catalectic," so-called "asystole." The process is perhaps most common in the lungs, in which case the source of the peccant particle is the recesses of one or other of the right chambers. The symptoms of embolism will be considered according to the organ affected.

Pulmonary Embolism.—The so-called "*hæmorrhagic infarct*" described under pathology is the lesion here,—"pulmonary apoplexy" is the old-fashioned name for the condition, an inappropriate one, however, as regards its etymology. The great *symptom* of the condition is *hæmoptysis*. The blood is generally pure, dark, and non-frothy, and the quantity varies much, but it never becomes so great as to necessitate the mere loss of blood being taken into consideration. With or preceding by a short interval the appearance of hæmoptysis, there is often some febrile disturbance, which, however, is usually neither great nor prolonged. On physical examination it is the rule to find no marked dulness over the site of infarction, the patch being usually small and isolated, but when there are several infarcts lying close together, and especially when inflammatory consolidation takes place around infarction, a considerable area of dulness may be produced. The most common auscultatory sign is the development of small bubbling sounds, the bubbles formed being, however, of a larger size than those that occasion the "vesicular bubbling" of Skoda or *true crepitation*, such as is heard in the incipient stage of pneumonia and in œdema of the lung substance. Bronchial breathing is exceptional, the breath-sound being usually "indeterminate" (Skoda) or essentially vesicular. Bronchial breathing implies, of course, that a large mass of consolidation has been produced, which is not often the case, and that the large bronchial tubes of the consolidated part are free for the passage downwards of the glottic breath-sound—a condition, one would think, often prevented by the presence of blood in them. The physical signs in typical cases are developed over a comparatively small area corresponding to the localisation of the infarct—a subject considered under pathology. When a large area, say the greater part of a lobe, is rendered dull, the presumption is that ordinary inflammatory consolidation has supervened around the infarct or infarcts, and the temperature is then apt to be maintained at a high elevation. When a large embolism occurs or several small ones occur in close proximity, pleuritic effusion is apt to result and mask the primary condition, although hæmoptysis will usually be present to indicate that the embolic process is in operation.

Cerebral Embolism.—A very important part in the symptomatology of cardiac disease is played by the embolic process involving the brain. Among the vessels, in which the embolus may be arrested, the middle cerebral artery of the left side stands pre-eminent. Its occlusion gives rise to right hemiplegia and usually more or less aphasia, the latter being sometimes very complete. Differences among cases depend essentially on the exact site of the lesion, and consequently the branches whose circulation is interrupted by the embolus. The "simple" mode of onset of hemiplegia, in which there is no loss of consciousness, is the rule, though occasionally the "epileptiform" and "apoplectiform" modes of onset are witnessed. The attack is remarkable among "strokes" in general, because of its frequent occurrence in the young; so much, indeed, is this the case that a "stroke" in a young person, especially of the female sex, at once suggests heart disease, and specially valve disease. Quite a large proportion of young female patients suffering from mitral stenosis become the subjects of such attacks of hemiplegia. The form of hemiplegia is not always the same, and the left side—the embolus being lodged in the right cerebral hemisphere—may be the affected one, as in a young girl lately under the writer's care. Moreover, owing to unusual vessels becoming plugged, instead of a definite hemiplegia resulting, quite irregular and anomalous manifestations of locally arrested cerebral circulation may arise, and these may be either trivial or grave and lethal. Thus the writer, when as a house-physician making one evening his usual round, while actually feeling the pulse of a patient suffering from cardiac disease, noticed the hand and forearm twitch convulsively, and the patient became suddenly sick, and immediately complained of violent headache. Next day there was still headache, and the head could not be raised from the pillow without sickness. Slight paresis of the left 6th nerve was noticed on the following day, but all the symptoms of the seizure had gone, and the ocular paresis disappeared a few days later. A fortnight after the same patient died from an embolus which evidently blocked a large vessel, violent headache in the occipital region, urgent vomiting, and speedy loss of consciousness being the symptoms produced, death resulting two hours after their sudden onset. Between these extremes all degrees of severity of seizure may be witnessed as the result of cerebral embolism, but as the special symptoms of the embolic process affecting the brain are considered in detail elsewhere in this work it is useless to refer to them at greater length here. Aneurysm or rupture (with fatal apoplexy) may be final results of embolism of cerebral vessels, and in regard to them the reader is referred to the section on pathology.

The writer has had no clinical experience of arterial embolism in the liver, while the condition of this organ generally in heart disease has been fully considered.

Embolism of the spleen produces occasionally swelling of the organ, accompanied by some degree of pyrexia and local discomfort or actual pain.

When the peritoneal covering becomes inflamed the pain may be acute, and a friction rub may be detected by stethoscope and hand. The clinical detection of splenic embolism is only likely to be accomplished when the infarction is large or multiple.

Embolism of the mesenteric arteries is accompanied, when the vessel blocked is large, by sudden abdominal pain followed by the indications of shock or collapse, and the later occurrence of profuse hæmorrhage per rectum. The accident is a rare one, and the writer has observed only two cases, in one of which the patient recovered. In the other the condition was proved post-mortem.

Large peripheral arteries—as the brachial, femoral, and popliteal vessels—are occasionally the seat of embolism, severe local pain ushering in the attack, and being followed by numbness and paresis of the limb, which becomes cold, pale, and finally livid. Aneurysm may ultimately result at the site of obstruction, owing to the necessarily impaired nutrition of the vascular wall at this spot.

There is usually little difficulty in finding the exact situation of a peripheral arterial plug. Gangrene of the part supplied may result, but in most cases a collateral circulation is easily established, the arteries of the part being sound.

In the foregoing account of the symptoms of cardiac disease in general, these are regarded as the product of that condition of circulation which we have designated "venous stasis," characterised by excess of blood-pressure on the venous or "return" side of the circulation. It must be admitted, however, that clinical experience makes us acquainted with the fact that heart cases are met with in which "venous stasis" is conspicuous by its absence, to use the hackneyed phrase, and this to the end of the case. Of the three cardinal symptoms the first—dyspnœa—under these circumstances, the writer believes, is never altogether absent. The sedentary man with failing heart, however, may never be aware of his disability, because of his never putting his heart to the test before sudden fatal syncope occurs. The writer well remembers the sudden fatal syncope of a relative of his own, who had never manifested any of the ordinary symptoms of cardiac disability till instantaneously fatal syncope occurred utterly unexpectedly in the midst of tranquil occupation. Yet the writer had often noticed as a boy this same relative give now and again that peculiar sigh he has described as a rare form of dyspnœa. Again, in the case of a friend, while apparently in perfect health, he often noticed this same occasional sigh for years before more ordinary symptoms of cardiac failure made their appearance, and the subject of them died in middle life after a short illness in which neither murmur nor dropsy was present, and in which the expression of the cardiac failure appeared on the arterial rather than on the venous side of the circulation, if one may so say. But even such cases bear out the belief in the paramount importance of respiratory interference in some form in all cases of cardiac failure. The writer has no doubt whatsoever that had the individuals whose cases he has referred to been subjected to effort their "wind" would have failed in the ordinary manner of cardiac dyspnœa. While admitting, as he must, the occurrence of such unusual cases as those related, he is firmly of opinion that had the patients survived longer venous stasis with its ordinary manifestations would have supervened. This conviction is derived from his observation of patients who first of all developed the symptoms of failure on the arterial side of the circulation, but later—often, indeed, only shortly before the end—the ordinary ones on its venous side. The two conditions that are specially apt to furnish examples of failure on the arterial side are aortic incompetence and fatty heart. The consideration of angina pectoris is excluded from this article. The curious condition known as bradycardia, which is usually associated with degeneration of the cardiac muscle, but which may complicate valve lesions, as mitral stenosis, may be regarded as a third. The comparatively slow pulse of aortic stenosis probably belongs to a different category, and within limits may be a physiological adaptation. Congenital bradycardia, too, must be regarded apart. Failure of the cerebral circulation is the striking feature of these rare cases in which manifestations occur on the arterial side—syncope, epilepsy, and distressing "cerebral" sickness being common among them. These symptoms are best developed in cases of simple muscle failure. Patients with aortic incompetence, again, may have extremely developed physical signs of their disease, and suffer much from dyspnœa and palpitation, without any development of dropsy or engorged liver, and they may go on in this condition for years. But even when this has been the case, the two lacking cardinal symptoms may become developed with great rapidity and in great intensity during the last few days of life. Between these two conditions an intermediate one may be quite unexpectedly produced, the features of which will be best described by an illustration. A man in early middle life, who had free aortic regurgitation, the result apparently of strain, and suffered severely from dyspnœa on exertion and anginal seizures on any effort, though he had never had dropsy or engorged liver, went to his work as usual one morning. Soon after he had commenced his work severe dyspnœa set in, and in spite of rest and careful treatment from the outset of the attack (it occurred in hospital, the patient being an employé), became more and more distressing, while signs of congestion of the lungs developed, with physical signs, for the first time, and the patient died towards evening, the post-mortem revealing, besides the aortic lesion and a dilated left ventricle, intense congestion of the lungs. Who can doubt that had this patient survived longer the lacking cardinal symptoms —dropsy and engorged liver—would have been speedily produced, and thus the case would have been brought into the ordinary category of chronic heart disease? The writer, in a case of aortic incompetence, was once struck, on feeling the patient's pulse, with the extraordinary length of intermissions that were recurring from time to time, and a few hours later the patient quite suddenly died, without any noticeable alteration of his condition. Can we doubt that an intermission, during which the relaxed ventricle would be exposed to the forcible rush of blood resulting from arterial recoil, over-balanced itself, so to speak, in such a way that it was unable to recover? And if this may happen in aortic incompetence, may it not also happen in cases of simple muscle failure, in which an intermission has tarried too long? Nay, even in the healthy heart may not the physiological intermission of cerebral inhibition occasionally pass beyond the point from which recovery can take place?

Lastly, the justification for the old view of sudden death from spasm of the heart must be considered. After death in cases of the kind the cavities of the ventricles are found obliterated, except the small supra-papillary space which contains blood. This occurrence cannot, however, be definitely associated with sudden death, after which flaccid ventricles are commonly found, and it may be that the tetanic contraction of the ventricles occurs only after their paralysis has brought the circulation to a standstill and the organism to somatic death. The one condition is quite as lethal as the other, and it is obvious that their effects will be practically the same as far as the circulation is concerned.

It is a well-known fact that, in a disease usually regarded with so little apprehension as anæmia, sudden fatal syncope may occur. Nay, even when the heart, as far as we know, is in perfect health, profound nervous impressions may induce fatal syncope, for the cause of which the most skilled and careful pathologist will look in vain, and such cases have occurred in young subjects. In most cases, however, of *apparently* appallingly sudden death, clear indications of cardiac failure have been present for a considerable time, though probably known only to the patient, who, ignorant of their significance, gave them no heed. When a man drops down dead in an assembly or in the street, it will often be that the transition from health to death has really been a very much longer process than it often is in pneumonia or malignant scarlet fever. It is, indeed, the latency of the disease rather than the suddenness of its termination that is remarkable. Rupture of the walls of a flabby heart or of a cardiac aneurysm are of course well-known causes of sudden fatal syncope, and it may be well to note that convulsion, from suddenly produced cerebral anæmia, is a common manifestation of the sudden failure of the heart's action in cases of the kind. Angina pectoris, with which we have nothing to do here, is a subject closely related with that of sudden fatal syncope, and in the writer's belief has its anatomical basis in disease obstructing the coronary vessels.

These brief remarks must suffice to indicate the few cases of heart disease that prove fatal without the development of venous stasis, a condition which would seem only to have been anticipated, and thus to have been given no opportunity for development.

A note may here be made of rare cases of mitral stenosis that prove fatal from bronchitis and engorgement of the lungs without the development of dropsy. The liver under the circumstances usually affords indications of venous stasis.

MALIGNANT ENDOCARDITIS

A short account must be given of the symptomatology of septic endocarditis.[1] This remarkable disease presents itself to clinical observation with symptoms belonging to an entirely different category from that to which the symptoms later described belong. The element of *venous stasis* we found to predominate in the production of most of the latter, and for those that remained the *embolic process* was largely responsible. But septic endocarditis commonly presents itself under a clinical aspect that bears little or no resemblance to that of ordinary heart cases. Constitutional disturbance of the usual febrile type is early manifested, and generally brings the patient to rest in his bed before any marked degree of even the first of the cardinal symptoms has developed. It is for such febrile suffering that the patient usually first of all seeks advice, and the evidence of cardiac disease is discovered only in the course of thorough systematic physical examination. The pronounced anæmia that is often early developed in the disease may indeed lead to even such physical evidence as there is being ignored—for instance, to the murmur of mitral or tricuspid incompetence being altogether attributed to the muscle failure of anæmia. This could only happen to the grossly careless observer, who remained ignorant of the daily rise of temperature, not to mention other symptoms. The spleen, as already mentioned, is enlarged in the great majority of cases of septic endocarditis, while it is only exceptionally so in ordinary heart cases.

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[1] For Etiology and Pathology, see p. 362.

"Typhoid" symptoms—dry tongue, delirium, subsultus, etc.—may be developed, but it is often surprising how long they remain absent in the presence of prolonged intermittent, remittent, or irregular pyrexia, the patient retaining a fairly clean, moist tongue, and some appetite, while his mind is entirely unclouded for months,—the writer has known a case last over a year,—in these respects the case resembling a tuberculous one. Albuminuria and hæmaturia, and the presence of casts in the urine, are of frequent occurrence. Petechiæ, with the development in some degree of a hæmorrhagic diathesis, are not rare. Hæmorrhages into the retina are apt to occur, and even optic neuritis has been observed on ophthalmoscopic examination. The occurrence of embolism in various organs is common, and the corresponding symptoms, so produced, help to make up the clinical picture of the disease, which latter, in spite of the views of pathologists as to its being capable of production by different micro-organisms, forms a distinct and, on the whole, well-defined clinical entity usually easy of recognition when its broad features are known. Aneurysms may develop at the seat of embolism and even in the case of large arteries—for instance, the femoral artery, as in one of the first cases seen by the writer. The cerebral hæmorrhage with apoplexy, that occasionally proves fatal to quite young subjects of the disease, is probably likewise associated with the embolic process.

In a large proportion of cases septic endocarditis complicates cases of ordinary chronic valve lesion—generally rheumatic—when the symptoms referred to become added on to those of such chronic valve lesion.

It has become customary to describe certain clinical types of the disease, although the advantage of such attempted classification is open to question.

(1) The septic type is characterised by the occurrence of rigors and perspirations, the "typhoid" state, occasionally more or less icterus, multiple arthritis, with or without pus formation, and hæmorrhagic and erythematous eruptions.

(2) The "typhoid" type resembles in its symptoms enteric fever with tympanites and diarrhœa, delirium, somnolence, or coma. In relation to this type the occurrence of infarcts in the intestines and of occasional ulceration of the bowels is noteworthy. It must be remembered that murmurs (systolic) arise not very rarely in cases of enteric fever independently of any endocarditis, and are probably associated with muscle failure of the heart.

(3) The "cardiac" type is that which usually has been preceded by rheumatic endocarditis and the lesions resulting therefrom. Indications of disturbed general circulation are more likely to be present than in other forms. In certain cases the two forms of endocarditis are difficult to differentiate. The presence of fever, especially in the evening, without the occurrence of arthritis, and otherwise unexplained, should suggest the possibility of the supervention of the septic disease. Enlargement of the spleen will increase apprehension in this direction, but the spleen is not enlarged in all cases of septic endocarditis, and may be temporarily enlarged from infarct in non-septic cases.

(4) The cerebral type is often associated with meningitis—cerebral or cerebro-spinal—but, as in the specific fevers, severe cerebral symptoms are apt to arise independently of gross lesion in the brain. A "comatose" form of septicæmia, apart from any endocarditic implication for instance, is well known (*vide* Fagge, vol. i. p. 587). Embolic hemiplegia and apoplexy from cerebral hæmorrhage, which is probably associated with the embolic process, form another "cerebral type."

With regard to *diagnosis* the septic endocarditis is specially apt to be confused with enteric fever, the two diseases having many symptoms in common. Widal's typhoid reaction now proves of the greatest value in distinguishing between them. The presence of hæmorrhages in the retina would afford evidence in favour of septic endocarditis.

Diagnosis.—A bacteriological examination, unfortunately, in this disease is usually of but little diagnostic help. Still, in every case it is most desirable that a thorough investigation of the blood should be made.

1. As pointed out in the pathological portion of our article, *leucocytosis* is generally well marked in cases of malignant endocarditis. This may prove of some diagnostic service in certain cases.

2. Many investigations have been made by numerous observers respecting the bacteriological characters of the blood, but with rather meagre results.

The blood is best obtained by the withdrawal with a hypodermic needle and syringe (used, of course, with the strictest antiseptic precautions) of some 5 to 10 c.c. from one of the superficial veins of the arm. This is then mixed with a suitably prepared and liquefied agar-agar culture medium, poured into Petri's dishes, and allowed to develop under proper conditions. By such an examination streptococci and staphylococci have occasionally been found. This method, however, has not proved satisfactory for diagnostic purposes in most cases, as usually the organisms are not freely circulating in the blood, and even when they are exceptionally met with occur in very small numbers.

3. It is interesting also to remember that Widal's reaction has been obtained in cases which have ultimately been found to be associated with the evidences of a malignant endocarditis, probably developing during typhoid fever, or possibly due to the bacillus typhosus.

Special Treatment.—Much of what is later said regarding the general management of cardiac affections is fully applicable to cases of malignant endocarditis.

Since, however, the disease is dependent on microbial invasion various measures have been suggested with a view (*a*) to destroy the organisms; (*b*) to lessen or annul the influence of their toxic products.

No medicinal agent is known which has been proved to directly destroy or arrest the development of the invading organisms. Various antiseptic and disinfectant drugs have been advocated, but their utility is very doubtful. Quinine, sulpho-carbolates, benzoates, salol and mercurial preparations have been used, and according to some with benefit in certain cases.

Arsenic has been strongly recommended, and is sometimes advantageously combined with quinine.

In a few cases hypodermic injections of pure yeast ferment have been administered.

Nuclein and nucleinic acids have also been advised.

All such agents, unfortunately, have proved quite inadequate in the majority of cases, and their use is not encouraged by what is known of the bacteriology of the disease.

The success of serum-therapy, however, in certain of the well-defined infective processes has suggested the application of such to malignant endocarditis. Hitherto the treatment has not always been conducted with the necessary scientific precision, and up to the present time the success has been very limited. Before any antitoxin can be administered with anything like bacteriological accuracy the exciting organisms must be detected. As indicated in the pathological section of this article, various different organisms are capable of setting up the conditions included under "malignant endocarditis." Unfortunately, in most instances, examination of the blood gives no help in ascertaining the particular organism present in the valves. Sometimes, however, when the primary infecting focus is known, or when an investigation of the infecting channel gives a strong indication of the probable organisms responsible for the endocarditic process, administration of the special antitoxin may not only be considered justifiable, but may prove serviceable. According to the nature of the specific irritant, antistreptococcic, antistaphylococcic, or antipneumococcic serums may be employed.

The possibility of mixed infection must be borne in mind, so that, for instance, if a case following pneumonia is not benefited by antipneumonic serum the administration of Marmorek's antistreptococcic serum may be justifiably tried.

PHYSICAL EXAMINATION OF THE HEART

THE PHYSICAL METHODS OF DIAGNOSIS.—*Inspection.*—In a suspected cardiac case the first point to be noted is the vascular condition of the neck. In nearly every cardiac case some evidence of abnormality in the circulation will be obtained on examination of this part: there will be exaggerated arterial pulsation, or there will be venous pulsation, while in aneurysm of the aorta, with aortic incompetence, there will likely be tracheal tugging when the chin is elevated. In many individuals free from cardiac disease in the ordinary sense there is some venous pulsation in the neck in the recumbent posture. On the other hand, there are certain peculiarly short-necked, stout individuals who even when suffering from cardiac disease give little evidence of visible pulsation in the neck, presumably because their vessels are so well hidden. In most cardiac cases, with enlargement of the left ventricle, the carotid pulsation is exaggerated in some degree so as to form a noticeable feature of the neck. It is, however, in cases of aortic incompetence that the visibleness of arterial pulsation reaches its greatest development, because in this disease there is the greatest variation between the maximum and minimum blood tension in the arteries. Increased visibleness of pulsation in the carotid is best observed in the upper part of the neck towards the angle of the jaw, while pulsation in the veins is best seen at the lower part of the neck. Venous pulsation in the neck while the patient's body is upright may be regarded as always abnormal, but it must be confessed that the circulatory disturbances need not be serious. For instance, most cases of chlorosis or anæmia show it. The pulsation is seen to be double, and a tracing of such pulsation taken with Dr. Mackenzie's phlebograph shows that there is indeed a double wave—a small and a large one. Till Dr. Mackenzie demonstrated the error o previous interpretations, it had been assumed that the small wave was th auricular wave, and the large wave the ventricular, while in reality th opposite is the case. A moment's consideration will show that Dr. Mackenzie's interpretation is the right one, apart from actual observation with the aid of instruments, by which the venous and arterial pulses of th patient are recorded simultaneously on a revolving cylinder. When w consider the relation of parts, the vein that is visible in the neck, th auricle and the ventricle, realise that the contraction of the auricle is immediately succeeded by the contraction of the ventricle, and remember tha between the vein and the ventricle the auricle is interposed, we have onl to ask ourselves the question: What is happening in the auricle during the first part of the ventricular systole? to be convinced that in the cervical vein during the first part of ventricular systole there must be a negative, not a positive wave, inasmuch as between the contracting right ventricle and the visible cervical vein is interposed the *expanding* right auricle, for immediately its contraction is over this last must expand and give rise to a negative wave in the veins of the neck. Not until the chamber has become again full will its contents transmit to the veins of the neck the ventricular impulse. It does happen in advanced disease of the heart that the ventricular contraction may be manifested by a positive wave in the veins of the neck from the outset of systole, but before this can occur the contractile power of the auricular walls must have ceased, and the chamber have become as it were a passive reservoir. The great majority of visible venous pulsations are double and of the auricular type described, although the venous pulse *may*, as stated, be alone represented by a ventricular wave occupying the whole period of the systole of the right ventricle. It is a remarkable fact that venous pulsation in the neck does not always coincide in frequency with the arterial pulsation: *extra* venous impulses being, as it were, interpolated from time to time. This was well seen in a case of bradycardia under the writer's care.

In many cardiac cases the most pronounced visible pulsation in the neck is that of the vein, as is often observed in mitral stenosis. In aortic incompetence venous pulsation may be absent, while arterial pulsation is apt to be exaggerated in an extraordinary degree. When in the latter disease, however, both kinds of pulsation are present, the inference is that the lesion on the left side of the heart has seriously interfered with the pulmonary circulation and led to much disturbance in the function of the right chambers. Sometimes other veins than those of the neck, as those of the chest, face, and upper extremities, are seen to pulsate, and the liver may pulsate in an expansile manner. Tracheal tugging is a sign of aneurysm of the aorta, and cannot be regarded as a sign of heart disease, though aneurysm frequently produces incompetence of the aortic valves: hence the necessity of reference to the sign here. Apart from true aneurysm, the arch of the aorta may be dilated, and thus its impulse be exaggerated, so as to be perceptible in the supra-sternal notch and at the sternal end of the right 2nd intercostal space. Deformity of the chest, however, may be the sole cause of such abnormal pulsations.

Capillary Pulsation.—In cases of aortic incompetence, on making a patch of erythema by rubbing the nail over the forehead, this phenomenon is developed—the redness deepening and paling with the pulse. Apart from aortic incompetence, this sign may be elicited in some degree of development under conditions of the pulse that resemble those characteristic of the lesion, for instance alcoholic muscle failure of the heart.

In examining the chest of a patient suspected of having heart disease the cardiac region, as a whole, must be carefully scrutinised. In young subjects of advanced heart disease the whole region is often prominent, owing to the enlarged organ opposing the effects of atmospheric pressure upon the framework of the thorax. This is especially noticeable when lung-expansion has been interfered with, and pigeon-breast has resulted. Again, the whole cardiac surface may seem to be thrust forward *en masse* during the cardiac systole, in which case the special visible areas of ordinary cardiac pulsation, to be described, cannot be distinguished, the condition being the result of a growth encroaching on the posterior mediastinum and pushing the heart forwards. It must be remembered that during diastole the heart is flaccid, while during systole it assumes a special shape, to accommodate which the parietes have to give way before the hardened mass of muscle the ventricles in systole represent. Even when a large aneurysm in the descending thoracic aorta projects the heart forwards against the chest wall, it is the pulsation of the heart itself of which we have chief evidence, though the two pulsations—that of the aneurysm and that of the heart—may be so intimately blended as to be indistinguishable. Apart from such rare conditions it will be found that the visible impulses of the heart are usually three in number, and rarely there is a fourth: (1) that of the apex situated normally in the 5th intercostal space, well within the left mammary line, and occupying a small area about that of a square inch. The apex-beat represents the contraction of the left ventricle, and when this chamber is enlarged the apex-beat is displaced downwards and to the left, and remains well defined, unless the ventricle becomes rounded in shape from dilatation, in which case the apex-beat loses its definition and often disappears altogether. Some individuals, though apparently free from heart disease, have no apex-beat. This has been explained in various ways, but the fact must be recognised. In effusion into the pericardium the apex-beat may be elevated, but it is difficult in such a case to be sure that the impulse taken for the apex-beat really represents it. (2) An ill-defined pulsation below the xiphoid cartilage is known as the "epigastric impulse," and indicates the movements of the right ventricle. Certain individuals, apparently free from heart disease, have marked epigastric impulse, presumably because of their having a short sternum. This impulse is present in nearly all cases of heart disease in which there is interference with the pulmonary circulation, and is apt to be specially pronounced in cases of pulmonary emphysema, in which there is not only obstruction in the lung-circulation and engorged liver, but a lowering in the position of the diaphragm as well. This impulse of the right ventricle must be carefully distinguished from actual expansile pulsation of the liver itself, which is an extreme result of obstruction in the pulmonary circulation, and which has been already referred to. (3) The third area of cardiac pulsation to be noticed is in the 2nd left intercostal space close to the sternum. Pulsation here is probably never normal, though it may be the left lung that is at fault and not the heart. This pulsation is usually associated with epigastric pulsation, and depends on enlargement of the infundibulum of the right ventricle. At one time such pulsation was attributed to the left auricle, the appendix of which comes to the front in this situation to the left of the pulmonary artery. But the appendix is often found plugged with clot, and even when active and dilated, gets as it were pushed aside by the dilated infundibulum. Paradoxical though it may seem, as good a case for the *right* auricular appendix being the seat of pulsation in the 2nd space to the left of the sternum may be made out, for it has actually been found greatly dilated and free from clot to the left of the sternum. Those who advocate the auricular origin of visible pulsation in the 2nd left interspace, admit that such pulsation is ventricular-systolic in rhythm, and account for it by a backward current into the left auricle from the left ventricle through the mitral valves, although the systolic murmur audible over the pulsation is quite absent from the apex, to which the downward directed valve curtains might be expected to direct it.

Pulsation in the 3rd left interspace is specially common, apart from mitral stenosis, in cases of chlorosis, in which the blood would seem to have difficulty in passing through the pulmonary circuit. These are just the cases in which visible venous pulsation in the neck is so manifest, bearing out the special implication of the right side of the heart in the circulatory disturbance. (4) In cases of great enlargement of the right chambers the *right auricle* occasions visible pulsation to the right of the lower half of the sternum.

Whenever the left ventricle is unduly exposed, whether it be from retraction of the left lung or from dilatation of the heart, it is common to see systolic retraction of the intercostal spaces *above* the apex-beat, a result simply of atmospheric pressure, the heart, of course, occupying less space during its systole than during its diastole. This must not be confused with retraction of the area corresponding to the left ventricle *including the apex*, which is a sign of complete pericardial adhesion, internal and external.

Palpation.—The apex-beat of the heart is the first object of attention when the hand is placed over the cardiac region. Valuable information is afforded in this way as to the condition of the most important chamber of the heart—the left ventricle—for the apex of the heart is formed alone by this chamber. The *situation*, the *extent*, and *force* of the apex-beat must in all cases when possible be carefully investigated,—that is to say, if the apex-beat is present, because in not a few cases of cardiac disease there is no apex-beat, and the negative fact is always to be noted, though it need not be given an undue share of attention, seeing that not a few individuals, who present no evidence of cardiac disease, do not possess an apex-beat. When there is an apex-beat its situation fixes at once the greatest extension of the left ventricle to the left, for the apex must always be the portion of the chamber lying to the extreme left. The extent of the impulse, which normally should occupy the space of about a square inch, again, is often increased considerably, as is specially noticeable in cases of aortic incompetence in which there is dilatation and hypertrophy of the left ventricle. A well-defined though extensive apex-beat may be taken as an indication that the ventricle still retains its form and has not become rounded or globular.

For reasons already referred to, absence of the apex-beat cannot be accepted implicitly as a sign of cardiac weakness, but if a previously present apex-beat ceases, it may be accepted as such a sign, and in corroboration of this inference it will commonly be noticed that the impulse of the right ventricle increases as that of the left declines, as was long ago observed by Stokes in typhus cases.

When the auscultatory sign, to be described later as the *bruit de galop*, is present, it is not very rare to feel over the apex a double shock during the cardiac diastole; if palpation precedes auscultation, it may be predicted in a case of the kind that the *bruit de galop* will be heard (*vide* "On a Rare Combination of Physical Signs," *Practitioner*, September 1896).

As a very rare occurrence in cases in which the pericardium is, internally and externally, universally adherent, there is no apex-beat proper, but in place of it there is during systole a general depression, *including* the region of the cardiac apex, followed by an impulse during diastole—apparently the result of the rebound of the relaxing and expanding heart. It is all-important to remember that there is no apex-beat present in the case referred to, so that this rare condition may not be confounded with the common inspection-sign of systolic recession *above* the apex-beat, that means only exposure of the heart, whether from cardiac enlargement or lung retraction.

To the observer with *tactus eruditus*, examination of the apex-beat by the hand is fraught with useful information in most cardiac cases. Apart from any accompanying thrill (to be considered later) its sharp, short stroke in mitral stenosis is most suggestive of this lesion, while in the hypertrophied heart of chronic Bright's disease the displaced, deliberate, slow heave is no less so.

Epigastric Impulse.—The impulse of the *right* ventricle is of an altogether different kind from that of the left ventricle, which is the apex-beat. It is a diffuse impulse, felt below the xiphoid cartilage, and appeals in most cases to inspection rather than palpation. Only in cases of great hypertrophy of the right ventricle does it become in any sense strong and "heaving," and in the latter case it is easily perceptible over the lower part of the sternum itself. In cases in which the right chambers of the heart are greatly hypertrophied and dilated, moreover, there is not infrequently expansile pulsation of the liver, so that there is apt to be much confusion of the latter pulsation with that of the right ventricle itself. The very rhythm of the impulse of the right ventricle has been and is still in dispute—some contending that it is diastolic and due to the inrush of blood. It is to be noted in this relation that the impulses of the two ventricles are seldom both pronounced at the same time. Again, in cases of great enlargement and hypertrophy of the left ventricle, with little or no implication of the right chambers, an abnormal impulse of the left ventricle may be perceptible in the epigastrium, and is then very liable to be taken for the impulse of the right ventricle. This happens specially in cases of aortic incompetence.

The impulse of the infundibulum of the right ventricle and that of the right auricle come under consideration as inspection- rather than palpation-signs, though they may be perceptible by means of the latter method. They have already been considered under inspection.

The shock resulting from the closure of the pulmonary semi-lunar valves is often perceptible on palpation in the appropriate area—the sternal end of third left cartilage — situated directly over the valves which lie very superficially. In cases of dilated aorta a similar impulse may be perceptible in the aortic area, which, however, be it remembered, is not situated over the valves, but at a distance from them so that the perception of such an aortic impulse is much less common.

Thrills which are perceptible by means of palpation are usually represented in auscultation by corresponding murmurs, with which in rhythm and other respects they correspond. Thus in cases of mitral stenosis there may be presystolic and diastolic thrills at the apex, which can sometimes more easily be separated by palpation than the corresponding murmurs by auscultation: the *crescendo* character and abrupt termination with the apex-beat of the presystolic, and the *diminuendo* character of the diastolic murmur, are easily recognisable by both methods, but a pause between the two is sometimes perceptible by palpation, that is absent in the case of auscultation. Evidently the later vibrations of the diastolic murmur in this case are imperceptible in palpation, as one can readily understand them to be. Diastolic thrill may be present at the apex in cases of free aortic regurgitation, when the history of the case and the usual vascular indications of the lesion will commonly save the observer from error with regard to the origin of the thrill. In excessively rare cases of simple cardiac dilatation, without aortic incompetence or mitral stenosis, a diastolic thrill has been felt, as a diastolic murmur has been heard (*vide Practitioner* vol. lii. p. 254, 1894).

Systolic thrill in the *aortic region* is common in cases of aortic stenosis. In the aortic region the systolic murmur that is commonly associated with dilatation of the arch may be accompanied by a similar thrill which the abnormal approximation of the vessel to the surface makes easily perceptible.

Occasionally a systolic thrill is present at the *apex* in cases of mitral stenosis accompanied by corresponding systolic murmur, which is usually at the time the only murmur present. Possibly, an apex systolic thrill may be felt in any case of mitral regurgitation with very loud murmur.

Percussion.—In the course of a systematic physical examination of the heart, it will often happen that inspection and palpation have furnished abundant evidence of the organ being enlarged before percussion is reached. But in not a few cases the estimation of the size of the heart must be essentially based on the percussion result alone, neither visible nor palpable impulse being produced, owing to the depressed vigour of the heart muscle and the alteration in the shape of the left ventricle, that is so apt to be associated with habitually incomplete systoles. Consideration of the heart *in situ* makes it at once evident on what principles the physical method of examination by percussion is founded. Except inferiorly the heart is surrounded by the resonant lungs, the anterior borders of which embrace the organ in such a way that a large portion of its anterior surface is covered by them. Moreover, these covering borders of the lungs are wedge-shaped, as they interpose themselves between the heart and the chest wall, that is to say, they become progressively thinner towards their edges. The anterior margins of the lungs separate at the level of the fourth cartilage, the border of the left lung passing outwards and downwards to a point situated over or close to the junction of the fifth rib with its cartilage. The border of the left lung then proceeds downwards and inwards along the sixth cartilage. Thus a more or less triangular space is formed in which the heart comes into direct contact with the chest wall, lung no longer intervening. The lower boundary of the heart, of course, corresponds to the upper boundary of the liver, the two non-resonant organs being indistinguishable by percussion. If, however, we define the upper boundary of the liver dulness to the right of the sternum, taking care to get beyond the right border of the heart—the right auricle—and from the level of the hepatic upper boundary draw a line, slightly sloping downwards, across the chest to the left anterior axillary line, such line will give an approximate representation of the lower boundary of the heart, a fact that is often readily demonstrated by the apex-beat being found immediately above it.

There are three directions in which it is necessary to determine the boundaries of the heart by percussion in order to estimate its size and ascertain its shape for practical purposes. From the size and shape of the dull area we infer the relative condition of the cavities of the heart. It is, of course, evident that the accumulation of fluid in the pericardium or pleuræ would render the estimation of the size of the heart by percussion impossible for the time being. The position of the right border of the heart should be ascertained above the upper boundary of hepatic dulness by percussing—say an inch above this boundary—from the right mammary line towards the sternum, and noting the first decided diminution of volume in the lung resonance that occurs as the sternum is approached. It is only when the right auricle has become so hugely distended as to be uncovered by lung to the right of the sternum that actual *dulness* is obtained on percussion of this region. Anatomically we know that the right auricle in health passes about half an inch to the right of the sternum, thus fully an inch from the median line, but it is covered by such a thick layer of lung here that its presence fails to modify the pulmonary percussion sound. When, on the other hand, we find absolute dulness to the right of the sternum, we are entitled to conclude that the right auricle is greatly distended and probably paralysed, while the degree of its distension is an excellent indication of the amount of obstruction in the pulmonary circulation. When there is no absolute dulness to the right of the sternum, but only a diminution in fulness or a certain "emptiness" of the resonance, as is indeed the common condition in all but most grave cases of heart disease, it is more difficult to fix the exact spot at which the presence of the auricle first modifies the pulmonary resonance, and this difficulty is increased by the approximation of the cartilages as they join the sternum, especially in certain individuals, exerting an influence on the percussion sound. Nevertheless by careful percussion towards the middle line, above the hepatic dulness, the point to which the right border of the heart extends can usually be ascertained with a fair amount of precision. Percussion up to the middle line, that is to say, over the sternum, is unsatisfactory, inasmuch as this bone has a peculiar (so-called osteal) resonance of its own, which interferes with the determination of the percussion sound that would otherwise be yielded by the underlying structures.

The next determination to be made is the greatest extent of cardiac dulness to the left of the sternum. A moment's consideration will make it plain that the portion of the heart that extends farthest to the left of the sternum must be the most important chamber of all—the left ventricle—and that its extremity represents the apex of the whole organ. Moreover, the greatest extension of the heart to the left will necessarily, owing to the shape of the organ, be at its lowest part in the vertical line of the body. When, then, percussion is made just above the level of the inferior border of the heart and from the left anterior axillary line towards the sternum, the first part of the heart that will modify the pulmonary percussion sound will necessarily be the apex, formed by the left ventricle alone; and, therefore, the greatest extension of the cardiac dulness to the left will represent the size of the left ventricle. When the apex-beat is discernible, percussion can be made directly towards it from the left anterior axillary line. Where there is no apex-beat the position of the apex must be approximately determined, as above described, by drawing a line across the front of the chest, from immediately above the upper limit of hepatic dulness to the left axillary line, almost horizontally, but sloping very slightly downwards. The first dulness met with in percussing from the left axilla towards the middle line just above this line represents the apex of the heart, and therefore of the left ventricle.

There remains to be determined the summit of the cardiac area of dulness. This determination should be made in the so-called parasternal line—a line drawn vertically downwards an inch to the left of the sternum Extension of the cardiac dulness upwards in this situation depend commonly on effusion into the pericardium (a condition not considered i this article), or on enlargement of the infundibulum of the right ventricle—conus arteriosus. Normally there should be no extension of the cardia dulness above the third cartilage. Of course, all these remarks presuppos air-containing lung parenchyma in the neighbourhood of the heart.

In noting the features of a cardiac case, the size and shape of the hea form most important points in the formation of the diagnosis. For a practical purposes measurements in the two directions indicated above, a the noting of the rib to which the cardiac dulness reaches upwards, afford perfectly sufficient statement of the size and shape of the heart. T results can be noted in a very brief yet perfectly satisfactory manner thu The costal cartilage to which the cardiac dulness reaches upwards in t left parasternal line is stated in Roman figures III. or II., as the case m be, and a line is drawn underneath, as under the numerator of a fraction. Underneath this line is placed, in Arabic figures, and to the observer's left, the distance stated in inches to which the right border of dulness extends to the right of the middle line, and to the observer's right the distance, stated in inches, to which the dulness extends to the left of the middle line at the level of the cardiac apex, which is of course the extremity of both the left ventricle and the whole heart. The note is made as if it were sketched in chalk on the patient's chest.

In the normal heart there is practically no dulness to be detected to the right of the sternum. Moreover, the difficulty of detecting a difference, in their percussion sound, between the right and left halves of the sternum, as it lies over the heart, is great and often insuperable. It suffices, then, to note any impairment of resonance there may be to the right of this bone, and to measure the extent of such impaired resonance in inches from the middle line at the level indicated. When any absolute dulness is found beyond the right sternal border it may be assumed at once that the right auricle is much enlarged. The writer has known such dulness to extend almost to the right mammary line, the right auricle being found post-mortem to have become a huge thin-walled sac, in all probability incapable of systole. Absolute dulness, of course, signifies that the distended chamber has displaced the right lung which normally overlies it. In ordinary heart cases the cardiac dulness to the right of the middle line varies from an inch to an inch and a half. It may be assumed that increase of dulness in the direction indicated implies impaired contractility on the part of the right auricle. To the left of the sternum, four inches from the middle line may be considered the maximum measurement of a normal heart, and the dulness will reach as far only exceptionally and in very large individuals. Usually in individuals with sound hearts the maximum measurement to the left of the middle line does not exceed 3½ inches.

Some examples of measurements in actual cases may be given in illustration of these considerations. The measurement $\frac{\text{II.}}{4\text{—}5}$, in a case of mitral stenosis, denoted great enlargement of the right auricle which had no doubt become little more than a simple reservoir, moderate enlargement of the left ventricle, and enlargement of the infundibulum of the right ventricle. The disproportionate enlargement of the right side of the heart is well shown by the figure above, and by that to the observer's left. The measurement $\frac{\text{III.}}{3\text{—}7}$ represented the cardiac dulness in a case of aortic incompetence late in the course of the disease, the right auricle having become much distended while the left ventricle was huge. The measurement $\frac{\text{III.}}{1\frac{1}{2}\text{—}4\frac{1}{2}}$ may be taken as representing the average size of the heart in cases of mitral stenosis, when admitted to hospital with engorgement of the right auricle. The distension of this chamber, in cases of moderate severity, rapidly subsides under rest and treatment, and the corresponding measurement of dulness to the right as rapidly diminishes.

Auscultation.—The evidence afforded by this method of physical examination is too often accorded a value greater than it merits, although it must be granted that in some cases it adds a precision to diagnosis unattainable by the other methods, and in all cases it gives the finishing touches to the diagnosis as far as that is based on physical signs. At the outset of this subject the student must be impressed with the necessity of studying the physiological sounds of the heart and such modifications of them as there may be, before he attends to the adventitious sounds present.

He cannot too soon realise the fact that many heart cases run their course from first to last without the development of any murmur. Among the variations that may be met with in the heart-sounds of apparently heart-healthy individuals there are many minor and, it is believed, unimportant ones. When the left ventricle is hypertrophied and thick-walled, the arterial tension being high, the first sound is often dull and toneless, while when the walls are thin and the cavity dilated, the arterial tension being low, the first sound is often loud and short so as to resemble the physiological second sound. It is similar shortening of the first sound that gives the heart-sounds in fever the resemblance to the heart-sounds of the fœtus *in utero*, that was so well described by Stokes. In the gravest fevers, however, as in typhus, a further change is produced as the result of profound failure of the heart muscle, namely, disappearance of the first sound altogether. As an event of the greatest rarity the second sound may likewise cease, so that the patient would be reasonably thought to be dead but for the faint flicker of the radial or other pulse. When the ventricular contraction is abrupt and the arterial tension low, as in palpitation of nervous origin, the first sound, again, is usually loud and short. Sir William Broadbent has called attention to a condition of the heart-sounds which he regards as of peculiarly bad omen, and in which the first sound is short and immediately followed by the second sound. The first sound may be reduplicated in all degrees from that in which the double sound seems to give only a prolonged and somewhat blurred character to the first sound, to that in which there are clearly two first sounds separated from one another. *Turrup-dup* may be taken as representing phonetically such a reduplication as the latter along with the following second sound. Naturally, in all these cases it is over the ventricles that we expect the modifications mentioned to be best heard. In mitral stenosis the first sound is apt to undergo a remarkable change, which with quiet action of the heart is most significant of the lesion. Such change is commonly termed *accentuation*. The first sound becomes peculiarly loud, short, and sharp. When the presystolic murmur is present it is invariably accompanied by a first sound of this kind into which it runs, so that the two together form a combination of murmur and sound, accurately represented by the syllable "trupp" with an accentuated end, to which reference will again be made. The resemblance between a reduplicate first sound and the combination of murmur and sound referred to, may be close when the heart is acting quietly, but exciting the heart's action by exercise or otherwise usually at once brings out the accentuation of the first sound, if it does not also develop the murmur, and doubt is dissipated in the case of stenosis.

Of Murmurs.—The foregoing remarks suffice to show how much valuable information concerning the heart, both for diagnosis and prognosis, may be obtained from modifications of the physiological heart-sounds quite apart from the production of *murmurs*, as we term the adventitious sounds, that in no way can properly be regarded as physiological sounds however modified. For the most part murmurs may be described as "blowing" sounds. It is difficult to apply such a description, however, to the auricular systolic murmur, which is further *sui generis*, inasmuch as it is *crescendo* in character and runs into the accentuated first sound, which at once brings it to a close. Some murmurs assume musical quality, transiently or permanently, and others acquire a loudness that renders them audible not merely over the greater part of the patient's trunk, but actually at a varying distance from it. The quality and loudness of murmurs have not, however been sufficiently utilised, either for diagnosis or prognosis, to render a dis cussion of them desirable here. Moreover, the caprice occasionally displayed by murmurs, in regard to the attributes under consideration, makes the probability small of their ever becoming of much practical importance. In the case of every murmur heard over the heart it is necessary for the observer to note—

(i.) The rhythm auricular or ventricular systolic or ventricular diastolic —in accordance with the physiological act taking place in the chamber of the heart concerned. When either of the physiological sounds remains audible with a murmur it is of great value in the determination of the rhythm of the latter.

(ii.) *The exact spot at which the murmur is heard loudest*, or, as it is called, *position of maximum intensity*. It must be remembered, however, that this does not necessarily correspond to any of the four areas that are usually associated with the four orifices and their valves.

(iii.) *The direction or the conduction of the murmur over the surface of the chest.*

(i.) Rhythm.—At the base of the heart murmurs may assume two rhythms, ventricular systolic and ventricular diastolic; and at the apex and sometimes over the ventricles they may assume three rhythms—auricular systolic, ventricular systolic, and ventricular diastolic.

The basic or arterial murmurs being the simpler will be considered first of all. During the systole of the left ventricle blood is rushing through the aortic orifice, and if a murmur becomes audible during this time we may presume that the current of blood is in some way interfered with. The term "obstruction" is often used to denote such interference, but it is open to very great objection, seeing that the aortic orifice may even be larger than normal. For instance, in aortic dilatation a very loud systolic murmur is often audible in the aortic area, of which the explanation is that, large as is the orifice of the vessel, its channel is larger still, so that the current entering through the former has to spread out to occupy the channel beyond. This condition we know to be the cause of arterial murmurs in general, so that in health all we have to do in order to produce a murmur with the blood current is to narrow the channel of an artery by pressure with the stethoscope. With regard to the aortic orifice a systolic murmur is only too likely to be produced by a trivial lesion, which accomplishes as regards results on the circulation an altogether unimportant effect on the blood stream—it may be a tiny fibrous nodule or thickening of the lip of a cusp, it may be mere thickening and stiffening of a cusp that prevents its natural effacement before the blood current, and, possibly, it may be a projecting atheromatous patch in the channel of the vessel beyond the valves. On the other hand, there may be extreme narrowing of the orifice, yet such grave lesion may be revealed on auscultation by a murmur of the same rhythm as, and otherwise indistinguishable from the murmur produced at an actually enlarged orifice and at an orifice only nominally diminished. (We have been constrained to use the expression "at," but "beyond" would be more strictly correct.) Lastly, as if to shatter hopelessly the value of a systolic murmur as a sign of aortic disease, not to speak of obstruction, this same systolic murmur is often heard over the aorta when it is perfectly healthy, as far as we know, in cases of anæmia. Notwithstanding, under certain circumstances having relation to accompanying phenomena, the diagnosis of aortic stenosis may to a considerable extent be based on this murmur, but even when this is the case, it must be admitted that the murmur then only contributes to the basis of the diagnosis: it does not essentially constitute it.

As a matter of clinical experience a systolic murmur in the "pulmonary area"—over or just beyond the pulmonary semilunar valves—is common, and, as described in another place, the mode of origin of such a murmur has been hotly debated. This murmur has become in clinical medicine specially associated with anæmia, in which condition of ill-health the murmur is very common. Still the fact remains that cases of anæmia in a pronounced degree are from time to time met with in which there is no murmur in the pulmonary or other area. In all kinds of heart disease, again, a "pulmonary" systolic murmur is common, and in certain cases of muscle failure of the heart it may be the only murmur audible throughout the illness, as for instance in alcoholic muscle failure.

In long-standing cases of mitral stenosis and like conditions it is not very rare for the pulmonary artery to become atheromatous and to lose its elasticity, so that dilatation is very apt to ensue. Without atheromatous change, indeed, it is likely that the artery will become dilated after prolonged exposure to high blood-pressure, and this event may, as in the case of the aorta, give rise to systolic murmur.

Even in ordinary cases of anæmia it is conceivable that this notorious systolic murmur in the pulmonary area may be produced in this way, the condition of anæmia seemingly leading to a difficulty in the passage of the blood through the capillaries of the lungs. We know how asphyxia leads to engorgement of the right side of the heart, and it is obvious that interference with the entrance of oxygen into the air-passages, and a diminution in the number or capacity of the oxygen-carriers of the blood, must both occasion some degree of interference with the pulmonary circulation and raise the blood-pressure in the pulmonary artery. Clinicists have long recognised that a systolic murmur over the pulmonary artery is the least valuable cardiac murmur with which they are familiar. It commonly accompanies other murmurs—both those dependent on valve lesions and those dependent on simple muscle failure. In a first attack of rheumatic fever it is very commonly encountered in association with a mitral regurgitation murmur, and (a fact of some interest in relation to the theory above referred to) it is commonly also associated with a tricuspid murmur. In the alcoholic heart and other forms of muscle failure, again, it may be the only murmur present. Sometimes it is present although there is no indication of ill-health of any kind, when it may be conjectured to result from some peculiarity in the contour of the vessel—some odd bend or the like. The low value of this murmur in diagnosis is firmly established, whatever may be our ideas as to the mode of its production.

Incompetence of the aortic valves is revealed to us on auscultation by a very definite *diastolic* murmur well conducted, downwards and to the left, over the cardiac area. When any portion of the second sound is audible the murmur runs off directly from it; that is to say, there is never the intervention of a pause between the second sound and the murmur.

When the blood tension in the pulmonary artery is habitually very high, as in cases, for instance, of mitral stenosis and pulmonary emphysema, the vessel is apt to undergo dilatation whether there be atheroma or not, and, as in the case of the aorta, the orifice, as well as the channel of the vessel, may finally take part in the dilatation, so that the valves fail to completely close the orifice, temporarily or permanently. Thus a murmur of pulmonary incompetence may arise as a curiosity of clinical observation—the second sound from which it proceeds being always much accentuated. This murmur of high pressure in the pulmonary artery is only an example of the same process that occasions the much more common murmur occurring under similar circumstances in the aorta.

As regards their rhythm, *mitral* and *tricuspid* murmurs may be: (1) *Ventricular systolic;* (2) *Ventricular diastolic;* and (3) *Auricular systolic,* commonly called *presystolic.*

It will be convenient to consider the ventricular systolic murmur first of all. This murmur indicates incompetence of the mitral or tricuspid valves according to its localisation and position of maximum intensity. Incompetence of the tricuspid valves is a very common consequence of disease, of one kind or another, affecting the left chambers of the heart, whereby the blood-pressure in the pulmonary circuit is much raised. In comparatively a very few cases there is stenosis of both mitral and tricuspid orifices, but the left lesion is always far in advance of the right. In such cases it is common for the simple murmurs of tricuspid and mitral regurgitation to have been the only murmurs observed during life.

In the immense majority of cases of heart disease in which the murmur of tricuspid incompetence is found, there is no lesion of the tricuspid valves at all, and their incompetence is simply the result of muscle failure of the right ventricle.

When a systolic murmur is heard at the *apex* we assume that the mitral valves have become incompetent, though the possibility of the murmur of aortic stenosis being conducted to the apex is difficult to deny altogether. Taken practically, the question is not one of great importance, and, given aortic stenosis, sooner or later the mitral valves are likely to become incompetent. In all forms of muscle failure of the heart mitral incompetence is very apt to arise, and with it the murmur we are considering. Then there are the cases in which this murmur results from actual damage to the valve curtains, rendering them incompetent. Rheumatism and septic endocarditis are the usual causes of the damage, but the latter is by far the more destructive to the valve structures, though the disease is comparatively rare; while in rheumatism, which is common, the damage to the valve is apt to be much less in the first instance, although the chronic process set up very frequently eventuates in stenosis of the orifice. Lastly, when mitral stenosis is thoroughly established, the necessarily deformed curtains, very commonly indeed, are, either permanently or from time to time, incompetent, so that the murmur of mitral incompetence is the most common of all the murmurs met with in cases of stenosis. Thus it happens that when a systolic murmur is loudest at the apex of the heart we associate it at once with mitral incompetence, and we have to ask ourselves practically these three questions: (*a*) Are the valves healthy, and is their incompetence the result of muscle failure of the heart? (*b*) Is there structural damage of the valve curtains rendering them incompetent, but without stenosis of the orifice? (*c*) Is there stenosis of the orifice?

The next murmur, the rhythm of which we have to consider in relation to the mitral and tricuspid orifices, is the *diastolic.* During the diastole of the ventricles blood is passing through the auriculo-ventricular orifices, and the condition of these orifices that produces a diastolic murmur is stenosis. (This statement makes no account of the very rare occurrence of a diastolic murmur in simple cardiac dilatation alluded to in another part of this article, p. 424; *vide Practitioner,* vol. lii. p. 254.) Tricuspid stenosis is a rare lesion, and a diastolic murmur is only very exceptionally produced by it, so that we may direct our attention exclusively to the mitral diastolic murmur, which is a common sign in cases of mitral stenosis. The murmur is usually best heard at the apex, but is less strictly limited to this spot than is the murmur to be next considered, being often audible over the ventricles between the apex and the lower part of the sternum. In rare instances it is widely distributed over the cardiac surface, so that when, as is usually the case, there is an accompanying systolic murmur, the double murmur may be, as far as sound goes, indistinguishable from the familiar "double aortic" murmur of aortic incompetence (*vide Med. Chron.* (1896), vol. vi. p. 174). The diastolic murmur of mitral stenosis, unlike the murmur next to be described, is somewhat *diminuendo* in character. The current of blood through the constricted orifice producing this murmur has both a *vis a fronte* and a *vis a tergo* in its production: the expanding left ventricle and the blood-pressure in the pulmonary circuit derived from the contraction of the right ventricle.

(3) The last murmur we have to consider is the *auricular systolic or presystolic murmur* of auriculo-ventricular stenosis. Like the last murmur, it is much more frequently produced in the left side of the heart, though a tricuspid presystolic murmur does very rarely occur. In several respects, as well as in its rhythm, this murmur differs from all the other murmurs with which clinical experience has made us familiar. As regards its rhythm, it immediately precedes the first sound, thus occurring during the latter portion of ventricular diastole. It is *crescendo* in character, and it is brought abruptly to a close with the first sound (modified as we have found) *when the murmur is at its greatest intensity.* This combination of *crescendo* murmur and first sound lends itself well to the usually accepted view as to the mode of production of the murmur by the current of blood resulting from the contraction of the auricle, for we can imagine the auricle expelling the blood with increasing force as its capacity diminishes, and we can understand the abrupt cessation of murmur at the moment of its greatest loudness, for how can the thin-walled auricle contend for an instant against the mass of muscle composing the left ventricle? Evidently, directly the ventricle enters into contraction, the current of blood issuing from the constricted orifice must be brought to a standstill.

(ii.) *Situation of Maximum Intensity of Murmur.*—In our endeavour to determine the orifice at which a murmur is produced, the place at which it is heard loudest must always be ascertained.

As a matter of fact all the orifices of the heart lie close together. To use Dr. Walshe's statement, "A superficial area of half an inch will include a portion of all four sets of valves *in situ*; an area of about a quarter of an inch, a portion of all except the tricuspid." It is therefore impossible to assign a murmur to a certain orifice because it is heard loudest over that particular orifice, seeing that the other orifices are in such close proximity, but the less important orifices—those of the right side—lie superficially, and one of these—the pulmonary—is so close to the surface that the shock of the closure of its valves can often be felt, as we have already found, on palpation. The areas at which pulmonary and tricuspid murmurs are usually best heard are, accordingly and respectively, "the sternal extremity of the 3rd left cartilage" and "the lower part of the sternum and adjoining area to the left of the bone where the heart is uncovered by lung." We expect murmurs generated at these orifices to be heard loudest in the areas named, which practically overlie the orifices concerned. As will be explained, however, under "Conduction of Murmurs," certain objections may be raised to the selection of the areas in question. It is impossible, however, to find better ones.

The principle on which the areas assigned in auscultation to the more important aortic and mitral orifices are selected is quite different, and is that of the *isolation* of the murmurs at some distance from the orifices at which they are produced. The "aortic area" is the "sternal extremity of the 2nd right costal cartilage"; here the aorta comes close to the surface, but of the two murmurs that may be generated at its orifice the systolic has unquestionably a better chance of being conducted hither in full intensity than the diastolic murmur. Clinical experience, as well as theory, bears out this statement, for one is often surprised at hearing so little in the aortic area of an aortic diastolic murmur that is loud below. The "mitral area," again, corresponds to the apex of the heart as well as the apex of the left ventricle. At the cardiac apex, then, we have to do with this chamber only. In the case of the direct or obstructive mitral murmurs, we can well understand their free conduction in the direction of the current that produces them, but for the murmurs of incompetence the apex as the place of their isolation would, at first, seem ill-chosen. It is known, however, that the disposition of the valve curtains, as lips projecting in the opposite direction to the regurgitant current, is calculated to reflect the murmur of incompetence forwards to the apex (*vide* Fagge: Reynolds' *System of Medicine*, vol. iv. p. 630). The frequent presence of this same murmur of mitral incompetence at the back is, of course, readily explained by the posterior position of the left auricle into which the regurgitated stream must directly pass.

While it is desirable that special attention should always be given both to the cardiac sounds and to any murmurs that may be audible in these four areas, auscultatory examination of the heart must not be *limited* to them. The necessity for this injunction will be evident on consideration of the following paragraphs.

(iii.) *The Conduction of Murmurs.*—The most important murmurs—the aortic and the mitral—have fairly definite lines of conduction, and especially is this the case with respect to the regurgitation murmurs of both aortic and mitral origin. As regards the obstruction murmurs produced at these orifices, the aortic is carried on in the course of the circulation, as might have been anticipated, and may be audible over the upper back in the left vertebral groove; the mitral is likewise carried onwards in the course of the circulation to the cardiac apex, beyond which it only exceptionally spreads. The systolic murmur of mitral incompetence, especially when there is structural change of the valve curtains without stenosis or very free regurgitation from muscle failure, is apt to be very well conducted to the back. On the other hand, in cases of regurgitation through a stenosed orifice, the murmur is often not conducted to the back, only a first sound being audible there, and the same condition is the rule in cases of mitral incompetence from muscle failure, the exception to the rule being found in cases in which the incompetence apparently is unusually great for the time being. Murmurs that are inaudible at the back may be audible in the axilla as far as the mid-axillary line, or even beyond it, while those audible at the back are usually heard continuously, as the stethoscope is passed backwards, from the apex to the spine. In a considerable number of cases the murmur is not only audible, but loud at the back to the right of the spine. As in these cases there is often tricuspid regurgitation as well as mitral, some have supposed that the loud murmur to the right of the spine is tricuspid in origin, but this is very doubtful.

The murmur of aortic incompetence is conducted downwards and to the left, towards the apex. In many cases it is heard over all the cardiac area, in most cases it passes well down the sternum, and it may even be most distinct at, or actually limited to, the lower part of this bone. Feeble murmurs are not rarely located to the left of the sternum between the 3rd and 5th cartilages. It will be evident that these last two positions properly belong to tricuspid and pulmonary murmurs respectively, but as regards the former a tricuspid diastolic murmur is exceedingly rare, much more rare than the rare lesion it indicates—tricuspid stenosis—while pulmonary incompetence as an actual lesion is still more rare, so that these murmurs are little likely to occasion error in diagnosis. Moreover, in aortic incompetence, the vascular phenomena, referred to elsewhere, are generally characteristic of this lesion.

There are certain *noisy* murmurs of which conduction in definite directions can hardly be recognised, and which may indeed be audible at a distance from the patient's body. Aortic incompetence is specially likely to furnish examples of such murmurs, which may be heard all over the trunk and along the spine from occiput to sacrum, as well as at a distance from the body. How little variation as regards murmur can be attributed to changes in the lesion was well illustrated by a case of the writer's. A patient was admitted to the wards so intensely cyanotic that he was at once bled. On his entering the ward most careful auscultation failed to reveal any murmur, yet a day or two later the patient presented a systolic murmur of extraordinary loudness audible all over his chest, and even at a short distance from it. The patient had been the subject of acute rheumatism many years before, and on post-mortem examination, a considerable time after his admission to hospital, was found to have a greatly constricted mitral orifice without any trace of recent lesion. Similar caprice in the behaviour of a murmur is often witnessed in cases of aortic incompetence, in which a long-familiar murmur may disappear during febrile or other disturbance of the circulation, while it cannot for a moment be doubted, from the arterial condition, that regurgitation is taking place as freely as ever. Such cases supply a warning not to base a diagnosis of such and such condition of lesion on any peculiarity of its murmur as regards loudness, distribution, or quality. A curious point with regard to musical *quality* in the murmur of mitral incompetence is the frequent loss of this quality by the murmur audible at the back. If we draw no special inference from musical quality in a murmur this curious experience will not disturb us, but it does raise a doubt in one's mind as to the musical murmur at the apex and the non-musical murmur at the back being, as is usually believed, one and the same.

Having considered the facts to be ascertained by the physical method of diagnosis in the abstract, it will now be necessary to reconsider them as they occur in nature, grouped together in varying combinations, according to the form of disease that gives them origin.

The Physical Signs in Combination of the Different Forms of Heart Disease

We shall in this section deal, in the first instance, with one of the simplest of the valve lesions—*obstruction at the aortic orifice or aortic stenosis.* From its very nature, this lesion, which has usually been produced by rheumatic endocarditis, is of slow formation, and the left ventricle — the most important chamber of the heart — has plenty of time to accommodate itself to the adverse circumstances the lesion imposes on it. The difficulty must be encountered first of all in systole, and, so long as complete systole on the part of the left ventricle is maintained, the difficulty is limited to systole. Only when systole fails in its perfect accomplishment because of leakage through the mitral curtains as a result of muscle failure, or because of imperfect emptying of the ventricle as a consequence of this same muscle failure, does occasion for dilatation of the left ventricle arise. The importance of these considerations in regard to the physical state of the heart in aortic stenosis is immense, and by them we can understand how it happens that this lesion of all others has been notorious for the length of time it can be endured with comparative impunity. When once, however, muscle failure has set in, the gravity of the lesion is great. The slow pulse that is frequently noticed in cases of the kind perhaps helps in warding off the development of muscle failure, and may be regarded as a manifestation of the *vis medicatrix naturæ*. Coming to a systematic investigation of the physical signs met with in a case of aortic stenosis, our first inspection should be made of the *neck*, in which arterial pulsation tends to be less evident than in most other forms of heart disease, while the presence and degree of venous pulsation depend on the development of muscle failure of the heart as a whole—in other words, on the implication of the right chambers—in the circulatory disturbance. On inspecting the chest the apex-beat may or may not be discernible; its situation in the former case will determine the size of the left ventricle, while epigastric impulse points to involvement of the right ventricle in the results of the lesion. It has been held that aortic stenosis tends to diminish the apex-beat, and in some degree this may be the case, but the vigour of the muscle of the left ventricle as well as the shape of the chamber has always to be taken into account with regard to the apex-beat. Inasmuch as aortic incompetence is often associated in some degree with stenosis, it may be well to bear in mind that incompetence would seem to tend in the direction of exaggerating the apex-impulse. *Palpation* will usually confirm the observation made by inspection as regards arterial pulsation in the neck, and the force, extent, and situation of such cardiac impulse as there may be. The characters of the pulse are reserved for special comment later. Over the aortic area a very valuable palpation sign may often be elicited, namely, systolic thrill, which may be felt all over the course of the aorta, from its origin deep in the heart to the termination of its contiguity with the upper part of the sternum at the 2nd costal cartilage, no doubt transmitted in part of the vessel's course through the pulmonary artery and the conus arteriosus, while it may be felt on a finger, in the suprasternal notch, being thrust behind the manubrium sterni. It must be remembered that a similar systolic thrill may be met with under conditions the very opposite to those of stenosis, namely, cases of aortic dilatation, of which condition exaggeration of pulsation and the auscultatory condition to be described will usually afford ample evidence. It may be said that aortic stenosis militates against aortic dilatation, but the two conditions are not incompatible, as a matter of pathological experience. *Percussion* affords information as to the size and shape of the heart, and therefore as to the condition of its chambers in regard to dilatation. It need not be here referred to in detail.

Auscultation affords very valuable evidence as to the presence of aortic stenosis, though the murmur—systolic—associated with the lesion is of itself of little value, and for this reason, that under the opposite condition —that is to say, dilatation of the orifice—the same sign may be pronounced. Such dilatation invariably involves the first part of the arch, and therefore the second sound is intensified and accentuated, while in aortic stenosis the second sound is ill-developed, feeble, and toneless as a rule, and in many cases entirely absent, and this too without there being any diastolic murmur. It must also be remembered that diastolic murmur is not rarely associated with dilatation of the aorta and consequent accentuation of the second sound, with which character the incompetence of the valves does not usually appreciably interfere. It follows that if we meet with a case in which there is a systolic thrill and a loud, harsh systolic murmur, followed by a feeble second sound, or no second sound at all, in the aortic area, the probability of there being aortic stenosis is great. The presence of slight diastolic murmur will rather enhance than impair the value of this evidence. The history of an old rheumatic attack still further strengthens the evidence, as do also the minor physical facts elsewhere referred to, such as slow pulse, deficient carotid visibleness, etc.

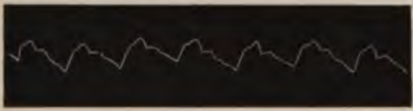

FIG. 1.—Anacrotic pulse of aortic stenosis.

The Pulse in Aortic Stenosis.—There are two varieties of pulse that have become associated in clinical medicine with aortic stenosis; probably neither possesses pathognomonic value in diagnosis, and to one of them greater diagnostic significance is to be attached than to the other. These are commonly known as the (1) anacrotic pulse and the (2) bisferiens pulse, and as regards their diagnostic value they stand in this order. The characters of these pulses are best studied graphically with the aid of the sphygmograph. (1) The sphygmogram of the anacrotic pulse is characterised by a sloping upstroke, which near the top is marked by a slight wave indicative of the position of the percussion wave, but the actual apex of the curve is above this, and is formed, not as normally by the percussion wave, but by the tidal wave, which is usually rounded and prolonged. The line of descent, like the upstroke, is sloping, and the dicrotic wave in its course is apt to be ill-marked. The pulse is usually infrequent, and has besides a sluggish wave-beat. When from nervous or other influence the pulse is rendered frequent, the characteristic features of the sphygmogram may become very much altered; on the other hand, a great degree of persistence of these same characters in spite of disturbing influences, as those of vaso-dilators and pyrexia, may be manifest.

When the anacrotic pulse is well developed and is associated with the characteristic murmur and thrill of the lesion, its diagnostic value is great. It must be remembered, however, that most pulses with a pronounced tidal wave are readily rendered anacrotic by the application of too great pressure with the instrument. The sensation imparted through the finger by an anacrotic pulse is sufficiently denoted by the sphygmogram. The infrequency and slowness of the pulse are its special features.

(2) The bisferiens pulse possesses very different features, and it certainly bears less definite relationship to the lesion than the anacrotic pulse. The upstroke is perpendicular, the apex of the percussion wave acute, and the cleft between this and the tidal wave remarkably deep, approaching in well-marked examples the "respiratory line," while the tidal wave itself is little less peculiar, its summit forming an acute angle while the aortic notch is situated low down in the line of descent of the curve. The dicrotic wave is usually, but not invariably, ill-developed.

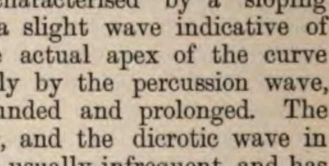

FIG. 2.—Bisferiens pulse of aortic stenosis. Valves incompetent.

This pulse can easily be recognised by palpation simply, at least the double beat can, though it may be impossible to distinguish the nature of the second beat in this way, that is to decide whether it is the tidal or the dicrotic wave that has become exaggerated. Exaggeration of the dicrotic wave in a case of aortic disease is, however, unlikely.

Aortic stenosis is often associated with some degree of incompetence of the valves, and this may be so developed as to modify the characteristic tracing of the more pronounced lesion.

Aortic Incompetence.—Incompetence of the aortic valves differs considerably from aortic stenosis with regard to its effect on the chamber of the heart, that is immediately and chiefly concerned—the left ventricle. The changes that occur in the latter are dilatation and hypertrophy. Very little consideration of the mode in which these changes are brought about will render it evident that the former process must be the first established, and there is no difficulty in accounting for the latter process, the former once established. With the advent of aortic incompetence, the ventricle has to accommodate the regurgitated blood *plus* the normal quantity, if the circulation is to be maintained. But the larger the cavity of the ventricle the greater must be, by physical law, the contractile power requisite to empty it. These theoretical considerations are abundantly borne out by clinical experience. It may be said, indeed, that aortic incompetence is the lesion of all others that is found associated with a huge left ventricle. As the muscle of this chamber begins to fail in vigour, secondary results of such failure become manifest, and take the usual forms of mitral regurgitation and imperfect systole, which in their train bring disturbance of the pulmonary circulation and engorgement, and finally dilatation of the right ventricle and auricle, usually associated with incompetence of the tricuspid valves and imperfect systole from muscle failure. Of course in a rheumatic case it is always exceedingly probable that the mitral valves are independently damaged and deformed, rheumatic endocarditis seemingly having a preference for the mitral valves.

Inspection.—One of the most notable signs of aortic incompetence is the exaggeration of arterial pulsation, which is perhaps nowhere better displayed than in the neck. The upper portion of the neck is the best to observe, in looking for this sign, as in the late stages of the disease venous pulsation may be pronounced as well, and the latter is most noticeable at the base of the neck. So long as the auricle retains its contractile activity the venous pulsation will appear to be double or undulating, as already explained, while the slightest pressure over the vein at the root of the neck at once stops the venous pulsation, the arterial pulsation remaining unaltered. The peculiar throb of the carotids at the upper part of the neck is one of the most striking clinical signs of this valve-lesion, and depends on the wide variation between the maximum and minimum blood-pressures that results from it. All the arteries of the body participate in the character, but the larger vessels are those to which attention should be directed. Very rarely various superficial veins may be seen to pulsate, but the same phenomenon is occasionally met with in other forms of cardiac disease.

Under inspection must be mentioned a minor sign of aortic incompetence already described, namely, the so-called "capillary pulsation" that becomes visible when a patch or streak of erythema is produced on the forehead by rubbing a hard substance, as the finger-nail, over the skin. The erythema is seen to redden and pale with the heart-beats.

Proceeding to the examination of the cardiac region the increased extent and displacement of the apex-beat will usually bear witness to the enlargement of the left ventricle. Epigastric impulse will be more or less pronounced according to the degree of implication of the right ventricle. In a few cases the impulse of a huge left ventricle would seem to be predominant even in the epigastrium, but in such cases the left ventricular impulse is widely diffused, while the indications of much implication of the right chambers are absent or ill-marked. Pulsation of the liver itself, again, is much less likely to be met with in aortic than in mitral cases. Our considerations led us to the conclusion that there is no lesion that more readily dilates the left ventricle than aortic incompetence, and when the ventricle is not only large, but has lost its shape and become rounded, a true apex-beat tends to disappear, and the impulse of the left ventricle that remains is diffuse and extensive. On the other hand, all visible impulse may cease, an indication of failing power on the part of the heart muscle, especially the portion forming the left ventricle. The differences with regard to the natural impulse that are met with in individuals must be borne in mind: what is referred to specially here is progressive change in the course of the case.

If the aorta becomes dilated, pulsation may become visible in the suprasternal notch, and in the second and first interspaces close to the sternum, a sign essentially due to the enlargement of the vessel, though promoted by the incompetence of the valves.

Palpation is useful chiefly to confirm the indications afforded by inspection. The force of the visible impulses can be better estimated by it, and so an inference be drawn more correctly as to the power of the heart muscle. Thrills are also ascertained by it. Over the aorta there may be a systolic or a diastolic thrill, or both. A systolic thrill—due to dilatation—is the more common in this region, though in rare cases a diastolic thrill is perceptible, and, curiously, may be limited to the apex. Such a thrill as this last probably in no way entitles to the assumption of any special condition of lesion in the valves, such as a long tag hanging down into the ventricle, etc., for the writer has found a diastolic thrill at the apex when the aortic incompetence was due to dilatation of the aorta and its orifice only, and was unassociated with any valve lesion—aortic or mitral. When the ventricular impulses, especially the left, are invisible, the *tactus eruditus* of a skilful observer may yet afford him information as to the power and size of the chambers.

Percussion.—By this method the usual preponderating increase in the size of the left ventricle can be demonstrated, the extension of cardiac dulness taking place towards the left—to the left with an inclination downwards more or less marked. That this should be so is evident from the anatomy of the heart. In cases in which the right chambers have become secondarily enlarged, the extension of the right auricle to the right of the sternum will enable the observer to gauge the dilatation suffered by this chamber and the degree of obstruction in the pulmonary circuit that has occasioned it. When dilatation of the aortic arch has reached a high degree, a "mediastinal" dulness—that is to say, a dull area passing across the middle line in the upper sternal region, and reaching to neither "costo-acromial" angle—may be detectable.

Auscultation.—The great sign that this physical method of diagnosis affords in aortic incompetence is a diastolic murmur, heard in some part of the triangle defined as follows: by a line drawn along the right border of the sternum from the second costal cartilage to the xiphoid cartilage, and by lines joining the extremities of this one with the apex of the left ventricle, which is, of course, the point of greatest extension of the whole heart to the left. Over all this triangle, or limited to any part of it, the diastolic murmur of aortic incompetence is audible. Most murmurs of aortic incompetence are well heard down the sternum and to the left of the lower half or more of the bone. Feeble murmurs, again, may be limited to a small area close to the left sternal border, lying between the third and fifth cartilages. Some aortic murmurs are transmitted specially to the apex, and these have to be carefully distinguished from certain mitral diastolic murmurs, the resemblance being increased by the possible association of both murmurs with an accompanying thrill. In all cases of suspected aortic incompetence it is necessary to explore the whole of the triangle defined above. The majority of cases of aortic incompetence also present a systolic murmur accompanying or replacing the first sound of the heart in the aortic area. This, as already emphasised, must not be accepted as a sign of aortic stenosis, inasmuch as it may be produced by the most trivial of projections on a valve or some little thickening or rigidity of the same, in which cases the obstruction is only nominal, or the same murmur may be met with, as already explained, when the orifice, far from being smaller than natural, is actually larger than normal, in dilatation of the arch. This last type of murmur may be not only very loud, but heard over a considerable area, the aortic arch being in contact with the chest wall to an abnormal extent. (Thrill may accompany such a murmur and be felt over an extensive area corresponding to the dilated vessel.) Valuable information as to the actual condition of the affected parts may be obtained from a consideration of the second sound of the heart when that is still present, while its absence probably indicates destruction or profound deformity of the valves. The alteration of the second sound that is of chief diagnostic importance is accentuation or intensification. The combination of a systolic murmur followed by such a second sound renders dilatation of the arch, involving the ascending portion, almost certain, and the preservation of the sound in this form indicates such a degree of integrity of the valves as to render it very probable that the incompetence depends rather on dilatation of the aortic orifice than upon valve change. On theoretical grounds it has been assumed by some that incompetence according to its degree interferes with accentuation of the second sound. Clinical experience fails to support such assumption, and one thing is absolutely certain: accentuation of the aortic second sound and diastolic murmur are frequently associated, the murmur following the second sound without the intervention of the slightest pause. Dr. Walshe long ago called attention to an alteration of the aortic second sound, that he conceived to be due to a short murmur preceding the second sound and running into it, as the mitral presystolic murmur runs into the first sound, and such alteration he no doubt correctly associated with dilatation of the aorta. The writer recognises the altered sound referred to, and the accuracy of Dr. Walshe's inference as to its pathological association, but he regards it rather as a modification of accentuation than as a combination of murmur with sound. Phonetically, the sign referred to is represented, according to Dr. Walshe, by the letters *phwi . . . tt.* In this relation it is curious to note that it is often difficult to distinguish simple accentuation of the first sound in mitral stenosis from the combination of short presystolic murmur running into an accentuated first sound.

The Pulse in Aortic Incompetence.—Long before the days of the sphygmograph the peculiar characters of the pulse in aortic incompetence had attracted the attention of physicians. Sir Dominic Corrigan in 1838 first described these charactors in relationship to the lesion, and throughout the medical world the pulse is known as "Corrigan's pulse." Perhaps the most concise statement with regard to its characters is that they consist of a very extensive and a very rapid transition from the maximum to the minimum blood-pressure. The term "the pulse of unfilled arteries" is objectionable, seeing that the arteries are never empty; the name "Corrigan's pulse" implies no theory, and does honour to one to whom honour is due. When the finger is laid on the pulse in aortic incompetence the stroke or "ictus" of the pulse is much exaggerated, not only by the rapidity of its onset, but by the shortness of its duration and its abrupt cessation. Thus the pulse is one moment full and strong, the next it has collapsed. These features are intensified by raising the patient's arm, which renders the collapse of the pulse more evident. If the front of the forearm is grasped by the observer's whole hand the pulse is easily felt up to the flexure of the elbow. The visibleness of the arterial pulsation has already been referred to (Inspection).

The sphygmogram possesses very different features from those it presents in cases of aortic stenosis. Only in one minor character do the sphygmograms usually agree, and that is in the tendency to ill-development of the dicrotic wave. The upstroke is perpendicular, the percussion wave exaggerated, and the line of descent precipitous. The tidal wave varies; sometimes it is rounded and sustained, while the dicrotic wave is deficient, and the line of descent slopes more than usually. This type of tracing the writer believes to point to dilatation of the aorta being the essential lesion, the incompetence being secondary in sequence as well as in importance. The other type of tidal wave is characterised by its summit forming an acute angle, as in the bisferiens pulse, although the cleft between this wave and the percussion wave is much less deep, while the tidal wave itself rises less high. It has been suggested that the two types of pulse tracing described may be diagnostic of the lesion commencing in the channel (dilatation) and in the valves of the aorta respectively, but the writer is satisfied that reliance cannot be placed on this diagnostic point.

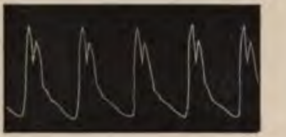

FIG. 3.—Typical pulse of aortic incompetence. Percussion wave well developed; dicrotic wave almost absent.

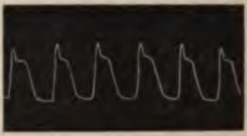

FIG. 4.—Typical pulse of aortic incompetence. Dicrotic wave absent.

Again, it is necessary to bear in mind that the lesions—aortic obstruction and incompetence—are apt to be associated in all varieties of proportion, and may be so equally developed that the tracing ceases in any way to be characteristic of either lesion.

Mitral Stenosis.—The physical signs of mitral stenosis:—*Inspection* reveals little that is distinctive of the lesion, unless it be in a few cases, the depth of cyanosis, such as is reached, leaving congenital disease out of consideration, only in one other common disease, namely, pulmonary emphysema. The muscular hypertrophy of the neck, and the labouring of respiration in this latter disease, are, however, likely to be so pronounced as at once to indicate the primary lung disease. In mitral stenosis venous pulsation in the neck is usually well developed, and along with it some exaggeration of arterial pulsation is commonly present. The apex-beat is often visible and fairly well defined, and displaced towards the left rather than downwards, while epigastric pulsation (R.V.) is usually evident.

Sometimes in severe cases a fulness due to the enlarged liver is easily discerned on inspection, and is seen to undergo expansile pulsation. Such pulsation must be distinguished from the ordinary form of epigastric pulsation resulting from the movements of the right ventricle enlarged and hypertrophied. Sometimes pulsation is visible above the third left cartilage, due to dilatation of the infundibulum of the right ventricle.

Palpation will usually confirm many of the observations made during inspection: for instance, the position and definition of the apex-beat, the exaggerated impulse of the right ventricle in the epigastrium, and—it may be—true pulsation of the liver. At the cardiac apex valuable information may be obtained from the presence of *thrills*, which may be in rhythm, presystolic, diastolic, or systolic. The last is the rarest and of least value in diagnosis, and may partake of the caprice so characteristic of the auscultatory signs of mitral stenosis. The presystolic and diastolic thrills are commonly associated, and are easily distinguished with the hand, the former running up to the apex-beat with which it ceases, the latter following the apex-beat after a short interval, while it often runs into the presystolic thrill, which, however, is easily distinguished by a fresh and increasing accession of strength until it is abruptly cut short by the apex-beat. The thrills—presystolic and diastolic—described are often quite as characteristic of the lesion as the corresponding murmurs. The shock of the pulmonary semilunar valve closure is often perceptible on palpation.

Percussion will indicate approximately the size and shape of the heart. As preponderance of the right chambers is common, increase of dulness to the right of the sternum, representing dilatation of the right auricle, is the rule; the right ventricle, however, occasionally directly modifies the percussible outline of the heart in a curious way, for the change is noticed to the *left* of the sternum; in long-standing cases the conus arteriosus or infundibulum becoming greatly enlarged, so that the origin of the pulmonary artery is elevated, and the cardiac dulness passes above the third left cartilage. Enlargement, to some extent, of the left ventricle is common, as a matter of experience, in cases of mitral stenosis. Exceptionally, however, it must be admitted one finds that percussion indicates the large right chambers and small left ventricle, that theory would lead one to regard as characteristic of the lesion. When in a long-standing case of mitral stenosis seen for the first time, enlargement of the cardiac dulness in the three directions—upwards, to the right, and to the left—may cause the dull area to resemble in shape and size the dull area due to pericardial effusion, as Sir Wm. Gull long ago pointed out. The average size of the heart in fifty-five cases of mitral stenosis was $\frac{\text{III.}}{1\frac{1}{2}—4\frac{1}{2}}$.

Auscultation.—The cardiac sounds always to be given precedence, in relation with murmurs, in the auscultatory examination of the heart often furnish evidence of chief importance in cases of mitral stenosis. The first sound at the apex is apt to be abrupt, accentuated, and short, and often possesses these characters quite apart from association with the presystolic murmur, of which it must be admitted such a modified first sound forms an integral part. The second sound in the same situation—the "apex-beat"—likewise often undergoes modification, and in one or other, it may be said, of opposite directions, for it may be doubled or it may be absent. In the former case it may well be doubted if the nomenclature is correct, and if we are dealing with the physiological second sound of the heart at all. The second sound of the pulmonary artery is usually, apart from reduplication, accentuated, the closure of the valves being often so forcibly effected as to communicate a perceptible shock to the palpating hand placed over the valves which lie superficially. The reduplication of the second sound (so-called) in mitral stenosis is often best heard to the left of the lower half of the sternum, and the relationship of such a reduplicated diastolic sound to the like sound met with in combination as the *bruit de galop* in muscle failure is of great interest. It rarely happens that in a case of mitral stenosis the heart sounds are normal in the absence of murmur, though for a time the lesion may thus be quite latent.

The most common murmur present in cases of mitral stenosis is one that has no *direct* association with the lesion at all, namely, the systolic murmur of mitral incompetence, but the very fact of the presence of stenosis implies that the mitral curtains are deformed, and it is hardly to be wondered at, if the murmur significant of incompetence should be—as it unquestionably is—the most common. This murmur may replace the first sound altogether, or it may follow the first sound, modified or normal. In either case, auscultation at the back, about the region of the angle of the left scapula, may afford a certain amount of evidence in favour of the lesion, by showing that the first *sound* is distinct in this region, while the *murmur* is absent. Unfortunately, the murmur being well heard at the back, and the sound being quite inaudible there,—whether present or not at the apex,—will not enable us to exclude mitral stenosis from the diagnosis. Moreover, in cases of mitral incompetence due to simple muscle failure, the non-conduction of the murmur to the back, and the presence of the first sound at the back, are as common as in mitral stenosis.

In a statistical inquiry as to the frequency of the different murmurs in a group of sixty cases, it was found that systolic murmur was present at the apex in 75 per cent of the cases, and that this murmur was inaudible at the back in 46·66 per cent, while it was present at both apex and back in 28·33 per cent (*Medical Chronicle*, vol. iii. p. 409, 1895).

Presystolic Murmur.—In the whole wide range of cardiac auscultation, the murmur that at once has the greatest diagnostic value, and shows the greatest caprice as regards its presence or absence at different times, is the notorious auricular systolic or presystolic mitral murmur. No other murmur, besides, has such definite characters, and no other murmur depends on such an unalterable condition of lesion. The presystolic murmur has usually a definite and limited localisation—the "apex-beat"—and it is only exceptionally heard widely distributed over the cardiac area, and only very rarely is it heard at the back. The accentuated first sound, however, which may be regarded as an integral part of the murmur, is often widely conducted. As regards its special characters the presystolic murmur is absolutely different from any other murmur known in clinical medicine. It is *crescendo*, and closes abruptly at its maximum with the modified first sound referred to. The coarseness of its vibrations gives it another peculiarity, so that it is the most palpable of murmurs, while the tactile sensations it occasions are similar to the aural, in that the crescendo character and the abrupt termination, when it is at its maximum, are as pronounced in the one case as in the other. The characters referred to are so peculiar that one can hardly help associating them with the commonly accepted view of the mode of causation of the murmur by the current of blood passing through the constricted orifice by virtue of the contraction o the left auricle, which must gather force as the auricle diminishes in capacity—contracts down on its contents—and must be brought instantly to a standstill the moment the left ventricle passes into contraction.

In the statistical inquiry already referred to, the presystolic murmur occurred in 53·3 per cent of the cases; that is to say, a presystolic murmur had been heard at one time or another while the patient was under observation in this percentage of cases.

Diastolic Murmurs.—A considerably more frequent murmur in mitral stenosis is the diastolic, but its diagnostic value is much less, inasmuch as aortic murmurs of the same rhythm are commonly well heard in the same area, and cases of simple dilatation of the heart presenting such murmur without any valve lesion are known, though very exceptionally, to occur. Even when mitral stenosis exists, and there is aortic incompetence present as well, it may be a matter of impossibility to apportion the diastolic murmur heard at the apex and over the ventricles to each of the lesions, for the diastolic murmur of mitral stenosis is much wider in its possible distribution than the presystolic, whose wide propagation is altogether exceptional.

Admitting, as we must, that aortic incompetence is frequently associated with mitral stenosis, and that its murmur is apt to be confused with the diastolic murmur of the latter, that the distribution of the two murmurs may not distinguish them, and that a diastolic murmur may be present in the mitral area when there is neither the one lesion nor the other present, our difficulties with regard to the significance of diastolic murmur are not yet at an end, although what remains of them concerns essentially the recognition or rejection of a possibly associated aortic incompetence.

The diastolic murmur that has yet to be considered has its place of distribution to the left of the sternum between the third and fourth or fifth cartilages, and is usually soft and blowing, while it immediately follows a greatly accentuated second sound. This murmur, which we have called "the murmur of high pressure in the pulmonary artery," unquestionably occurs in cases in which there has long been excessive pressure in that artery, so that its dilatation is accomplished and incompetence of its valves results, in the same way as aortic incompetence results from like changes in the aorta. In no lesion is the blood-pressure in the pulmonary artery apt to be long maintained at such a degree as in mitral stenosis, and hence the frequency of occurrence of this murmur in cases of the lesion.

At times the true diastolic murmur of mitral stenosis is practically indistinguishable from that of aortic incompetence, and is preceded by the systolic murmurs of coexisting mitral incompetence, etc., so that a double to-and-fro systolic and diastolic murmur is heard over the whole heart. In one case of the writer's, the patient came into hospital with a systolic murmur only, but later developed a loud and prolonged diastolic murmur which was widely propagated all over the cardiac surface. This murmur, along with the accompanying systolic one, closely simulated the ordinary "double aortic" murmur of aortic incompetence, except in one curious and interesting respect, that it did not seem to run off directly from the second sound, where that sound could be heard along with the murmurs towards the base of the heart. Difficult as it is to explain the intervention of an appreciable pause or interval between the second sound and the diastolic murmur of mitral stenosis, it seems impossible to understand how a pause could occur between the sound and following murmur, when the latter is due to incompetence of the arterial valves—aortic or pulmonary.

The Pulse in Mitral Stenosis.—Much discussion has taken place with regard to the state of the pulse in mitral stenosis, some observers contending that it is characterised by perfect regularity and well-maintained—even high—arterial tension, others that irregularity is its great feature. The writer has carefully studied the subject and has arrived at the following conclusions:—(1) Regular pulses of both high and low tension are commonly met with, the former occurring in the earlier stages of the case, the latter towards the end of the case, but low tension may be assumed by the pulse in the early stages, determined by physiological individual peculiarity or by disturbing agencies, such as the presence of pyrexia. (2) The pulse that is most characteristic of mitral stenosis is an irregular one, in which some of the beats are of fairly high arterial tension, and others abortive and of quite low arterial tension; these two types of pulse or—speaking with reference to the sphygmogram—of curve occur in all degrees of proportion, and series of beats of one or other kind are commonly interrupted by a beat or more of the other type. One may, in fact, regard this stage as made up in varying proportion of the preceding and succeeding stages already referred to. It must be admitted, however, that an indistinguishable pulse is frequently met with in cases of simple muscle failure of the heart. (3) As the pulse was regular and of good tension in the first stage, so it may pass into a third stage in which it is again regular but of low tension.

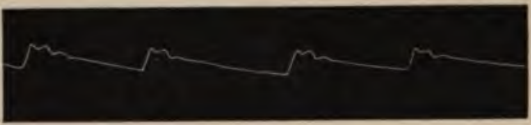

Fig. 5.—Typical pulse of mitral stenosis in first stage. Pulse regular and of good tension.

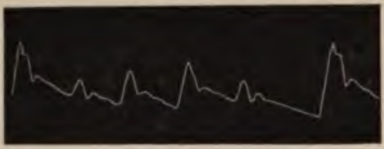

Fig. 6.—Typical pulse in mitral stenosis in second or irregular stage. The curves may be regarded as constituted of a mixture in varying proportion of those composing the pulse of the first and third stages.

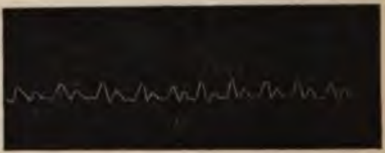

Fig. 7.—Pulse in mitral stenosis in third stage. Curves regular and of low tension.

The bigeminal pulse is not very rare in mitral stenosis, but is generally —not always—met with when the heart is under the influence of digitalis. It probably bears relation to the muscle of the heart rather than to the lesion.

There is no form of sphygmogram that, alone, enables the observer to make the diagnosis of mitral stenosis; the pulse may be regular and of high tension, it may be regular and of low tension, and it may be irregular and composed of both high and low tension beats, the latter being usually more or less abortive. Lastly, a patient quite early in the clinical evolution of the disease may have a low tension regular pulse under a disturbing influence, and as he recovers, his pulse may ultimately assume the characters of a high tension pulse, having previously passed through an irregular stage in which beats of both high and low type—the latter more or less abortive—occur side by side, though commonly the beats of one or other type appear in series, separated by the interpolation of one or more beats of the other type. Too much importance must not, therefore, be attached to the condition of pulse in mitral stenosis; it may be a "third stage" pulse when the patient is in no danger, and it may be a "first stage" regular pulse, *i.e.* of well-maintained tension, almost to the end, while the irregular or middle stage pulse is by itself of no gravity in prognosis.

Mitral Incompetence.—In no department of clinical medicine has a greater change in thought taken place during the last quarter of a century than in that concerning incompetence of the mitral valves. The importance of distinguishing *mitral disease* associated with *incompetence* and *mitral incompetence* independent of *mitral disease* at the beginning of that time was to a large extent, if not altogether, ignored. For such a state of thought the responsibility may be attributed to the ignoring of two simple, self-evident facts: (1) That the mitral curtains were never constructed to act independently of the contractile heart muscle, so that whenever the latter fails in its two obvious functions of diminishing the orifice to be closed by the valves and of supporting the curtains in action there must be a likelihood of incompetence resulting; and (2) That the great *lesion* of the mitral curtains found after death in the most common cases, the rheumatic, was not crippling of the valves, so that they could not close, but deformity of them resulting in narrowing of the orifice—in stenosis. It is not denied that lesions of the mitral curtains without stenosis are met with, amply sufficient to explain their incompetence alone, but it is affirmed that such cases are the exception and not the rule—it may be said the exception that proves the rule. Nay, in the most pronounced of them there are the best of reasons for believing that the damage of the structure of the curtains was not—even in these extreme cases—the *only* cause of the failure in the function of the curtains, but was inseparably associated in the accomplishment of such failure with weakening and debility of the heart muscle. Moreover, when stenosis of the mitral orifice was found after death in cases in which the clinical evidence for long had pointed to incompetence as the only condition, the obvious fact that whenever there is stenosis of the mitral orifice there must be deformity of the curtains, and consequently a likelihood of incompetence, was seized to do duty in explanation of the clinical experience. To put the matter shortly, the essential lesion (the stenosis) was ignored, and the mere accident of the lesion (the incompetence) was yielded a share of importance in disturbing the circulation to which it had absolutely no title. That the murmur of incompetence is by far the most common murmur in cases of mitral stenosis has been elsewhere demonstrated, but it is idle to say that the minute regurgitant current it probably denotes is the cause of the profound disturbance of the general circulation in mitral stenosis.

The cases associated with the most serious damage to the valve curtains, apart from stenosis, are those of septic endocarditis, in which the disturbance of the general circulation that must inevitably result is but a minor matter in the evolution of the case, seeing that hitherto the disease has proved inevitably fatal in an acute or at least subacute illness, which compels the patient to remain in bed (*vide* Septic Endocarditis, p. 393).

Skoda, it is believed, first called attention to the frequency of a systolic murmur in the "pulmonary area" in association with mitral incompetence and the consequent dilatation of the right side of the heart, and many years later the view (which we cannot regard other than as a great heresy) was advocated that this systolic murmur in the "pulmonary area" was nothing else than the murmur of mitral incompetence conducted to the surface at this spot by the left auricular appendix presumed to be dilated. In support of this theory a disposition was manifested to change the position of this long-familiar murmur, and move it outwards "half an inch or more to the left of the sternum" in the second intercostal space, to the spot corresponding to the tip of the left auricle as it lies to the left of the pulmonary artery. It was alleged that in chronic cardiac cases associated with habitual engorgement of the right side of the heart, the appendix of the left auricle becomes dilated, and to such an extent, indeed, as at times to occasion visible pulsation in the second interspace. Moreover, it was held that the murmur of mitral incompetence is frequently heard over such dilated appendix, conducted as if by predilection to this spot even when absent at the cardiac apex! The pulsation in the second left intercostal space referred to, used to be regarded as auricular systolic to fit in with this explanation of its being due to the auricular appendix, but as a matter of fact this rhythm of impulse could not be demonstrated—the impulse was obviously systolic. Worsted in their argument, the advocates of the auricle being the seat of the pulsation in the second left space shifted their ground, and contended that the pulsation and the murmur were alike due to a regurgitant current through the mitral orifice from ventricle to auricular appendix!—the deviousness of its course being completely ignored.

As a matter of fact, in all cases of cardiac disease in which the obstruction tells heavily on the pulmonary circulation, and in which the right chambers of the heart become enlarged, the right ventricle rather than the appendix of the left auricle it is that bears the brunt of the struggle and is the first to become enlarged. The advocates of the view we are considering would have had a less unfortunate case had they limited their theory to cases of mitral stenosis, in which the auricle is apt to be dilated, though its appendix may take no part in the change and be actually blocked with clot, but the great field of anæmic and other cardiac debilities was too tempting for them. Henceforth was not the notoriously common murmur of anæmia explained to their satisfaction? They had evidence of general cardiac disability in plenty, but why should such special disability fall thus to the share of the left auricle so as to account for its disproportionate dilatation in the absence of mitral stenosis? As we have affirmed, as a matter of fact post-mortem examination demonstrates, that when there is obstruction in the pulmonary circuit it is the right chambers of the heart that dilate, and that when there is obstruction at the mitral orifice the same chambers suffer the like change, though in common with the weak-walled left auricle, whose appendix suffers least, or even not at all. When, however, the right ventricle becomes dilated its infundibular portion is especially prone to give way, and as the conus dilates it tends to thrust aside the appendix of the auricle, while the position of this chamber proper is essentially a posterior one—very far back indeed. The pulsation attributed to the left auricular appendix is obviously the pulsation of the infundibulum, as was long ago shown by Dr. Mahomed. Then, again, whenever there is obstruction in the pulmonary circuit of such intensity and duration as to occasion pulsation in the second left interspace, close to the sternum, it is usually easy to feel with the hand the shock due to the too forcible closure of the semilunar valves, because they are here placed so superficially.

It will be asked why in the most common cases—the anæmic—the right chambers of the heart should specially suffer? The cause is probably that the morbid blood has difficulty in passing through the lungs; whether we cut short the supply of oxygen from the respiratory passages, or limit the capacity of the field for oxygenation, must not the result be similar? and if so, must not embarrassment of the right chambers be the first result—that of the left being often considerably postponed? In severe fevers in which the tendency of the blood to stagnate in the lungs is notorious, Stokes long ago called attention to the engorgement of the right ventricle, whose impulse grows apparently stronger as that of the left ventricle fails.

But though so well developed in cases of anæmia this systolic murmur in the pulmonary area is a not rare one in all kinds of cardiac disability, and in them too it may stand alone—the only murmur present in the whole cardiac area. The murmur, again, is not very rarely met with in the examination of candidates for assurance and for the public services, who seem to be in perfect health—possibly some anatomical abnormality of contour or course may be the explanation. We have already called attention to the dilatation of the pulmonary artery itself, that is met with in cases of long-standing mitral stenosis and other conditions resulting in congestion in the pulmonary circulation, and referred to the dilatation and actual atheromatous change that may ensue in a vessel so exposed to excessive pressure. There seems to be no reason why a systolic murmur should not be produced in the pulmonary artery under these circumstances, in the same way as under like circumstances it is produced in the aorta. Most of those who have been engaged in teaching clinical medicine have probably at one time or another met with this experience in the presence of an inquiring student:—A patient with a first attack of acute rheumatism develops a systolic murmur at the apex, but in a day or two there may be a similar murmur in the other areas, including the tricuspid and pulmonary; "Why," asks the inquiring student, "am I told that the same murmur which indicates disease of the valve in the mitral area has no such significance in the tricuspid and pulmonary areas?" The teacher will do well to base his reply on our accumulated clinical and pathological experience of facts, and not attempt a reply based on any theory. That such a murmur in the pulmonary area is common under the circumstance is unquestionable.

It must be admitted that as yet we have little knowledge of the mode of production of a systolic murmur in the pulmonary area, either standing alone or associated with murmurs elsewhere: it is probable, too, that the mode of production is not always one and the same. In a case of evident muscle failure of the heart—say due to alcoholism—this murmur may be the only murmur present, and it is difficult to deny its definite association with the muscle failure and consequent cardiac disability. No one will deny the element of muscle failure in most cases of anæmia. The murmur as an entirely isolated phenomenon, as met with, for instance, in apparently healthy candidates for the public services, is more difficult of interpretation. The practical rule, however, must be that a systolic murmur in the pulmonary area is the murmur of lowest value among all the murmurs with which clinical practice has made us familiar. Occurring in a case of evident muscle failure, it adds little information to that already acquired in other directions, but on the other hand one must not neglect the murmur altogether, seeing that it is not rarely associated with conditions of cardiac disability.

Mitral Incompetence as a Result of Muscle Failure.—One of the most common results of muscle failure of the heart—and this quite independently of its pathological cause—is mitral incompetence, that is in no way associated with any lesion of the valves. Mitral incompetence of this mechanism is met with under many morbid conditions, of which we will take only two as examples: the heart failure of anæmia and the heart failure of Bright's disease—the one curable, the other sooner or later lethal from concomitant circumstances, though capable of great and prolonged improvement from time to time during its course. The murmur of mitral incompetence present in the former cases was for long misinterpreted, while that present in the latter cases was for as long practically ignored, though the obvious signs of a failing heart were only too striking in the forms of dyspnœa, dropsy, and engorged liver, not to speak of the distension and pulsation of the veins of the neck and a host of minor indications.

Physiologists a long time ago recognised what they termed "the safety-valve" action of the tricuspid apparatus, by which term they denoted the fact that under excess of work to be done by the contraction of the right ventricle, the healthy tricuspid valves may permit regurgitation taking place, and so relieve for the moment embarrassment of the ventricle. It is known now that the same occurrence is common, and of much greater importance on the left side of the heart. When the arterial pressure is high, and the left ventricle labouring to maintain the circulation, it is now a familiar fact that the mitral valves often become incompetent, and that, too, in all probability for the immediate if not for the permanent benefit of the individual.

Muscle Failure of the Heart.—The consideration of mitral incompetence necessarily leads us on to the consideration of muscle failure of the heart in general, of which it is so often a secondary consequence. Probably in no case of mitral incompetence can it be said, however grave the structural damage to the curtains, that either the incompetence or the disturbance of the general circulation is solely the result of such structural damage, for invariably associated with this latter is an element of muscle failure that we cannot possibly afford to ignore. But there are cases of muscle failure that run their course and display all the ordinary indications of a failing circulation without ever developing, as far as we know, the condition of mitral incompetence. Of a silent mitral incompetence we know nothing, and considering the great frequency of the condition as denoted by its murmur, it seems likely that a silent regurgitation through the mitral curtains is a pure assumption having no basis in fact.

How then is to be explained the unquestionable occurrence of venous stasis and its usual effects as the result of muscle failure of the heart without any evidence of mitral incompetence from first to last of a long course? Evidently the explanation of the fact is amply provided for by the clearly established fact that an overburdened ventricle—be it right or left—has another mode of expressing its embarrassment than that obtained by the permission of incompetence of its auriculo-ventricular valves. The occurrence was long ago recognised (only an unfortunate name—asystole—naturally associated with most lethal significance, was given it, and the frequency of its establishment ignored), and consists of *the non-completion of systole.* In health the supra-papillary space of the ventricle, *i.e.* immediately below the valves, always contains blood at the end of systole, but with this exception the chamber is normally emptied. When there is muscle failure, however, residual blood may remain at the end of systole elsewhere than in the supra-papillary space. If any one has doubt as to the truth of this statement, let him look at the heart post-mortem in a case of muscle failure with huge rounded dilatation of the left ventricle, and ask himself how long he can suppose it is since such a ventricle completed its systole in the physiological way. To denote this condition of incomplete systole, on the advice of a scholarly friend, the writer has used the term *systole catalectic,* implying that the systole stops short of its completion.

One would naturally suppose that the same muscle failure that occasions systole catalectic would also occasion mitral incompetence, but as a matter of clinical experience this does not seem to be necessarily the case, though the two conditions are no doubt commonly coincident.

There are two auscultatory signs of very different nature, that are so commonly associated with muscle failure of the heart as evidently to bear definite relationship to it: one of these is so frequently met with under other circumstances — mitral valve disease and mitral stenosis — as to be little characteristic, the other we may consider as the *special sign of muscle failure* whether the latter be transient or permanent. The former is the murmur of regurgitation through the mitral and tricuspid orifices, owing to incompetence of the sound mitral and tricuspid valves, but this has been already referred to in sufficient detail.

Till lately the possibility of a diastolic murmur being produced in a simply dilated heart, the result of muscle failure, was usually emphatically denied, but of recent years evidence has been accumulating to show that a diastolic murmur of very considerable loudness (and actually accompanied by a thrill) may be produced in a simply dilated heart without any valve lesion whatsoever. In the face of this recent experience it is curious to find recorded in

Dr. Stokes' classical work on Heart Disease, published in 1854, a case of the kind. Two cases occurred in the Manchester Royal Infirmary a few years ago, and were recorded in the *Practitioner* for 1894. In these cases not only was there a diastolic murmur (accompanied by thrill), but there was also a reduplicate diastolic sound at the apex. During life, both cases had been regarded as cases of mitral stenosis, but neither this lesion nor aortic incompetence could be found post-mortem. It is sufficient here to point out the existence of such cases, however rare they may be. So seldom are they met with, that for practical purposes they scarcely impugn the usually accepted rule that a diastolic murmur means either arterial incompetence or auriculo-ventricular stenosis. They are of immense importance, however, in demonstrating to us how profoundly ignorant we are as to the mode of production of some of the signs with which we are well acquainted. The diastolic murmur referred to is of extreme rarity, but the *bruit de galop* (to be immediately described) is one of the most common of auscultatory signs, yet as to the mode of production of both these signs no satisfactory explanation has ever been given.

The auscultatory sign we are fain to regard as the great sign of muscle failure is a peculiar triple rhythm of (apparently) the cardiac *sounds*. The sign attracted the attention of French physicians long before it received from English authors the consideration it unquestionably merits, and so the French name has almost passed into our own language, and we call the sign the *bruit de galop*. Evidently one of the sounds is systolic and two are diastolic. Indeed, as already noted, there may be felt at the cardiac apex two distinct shocks or thuds that correspond with the diastolic sounds. We have, perhaps not without some clumsy writing, avoided the use of the term "reduplicated second sound," which might seem the natural one to employ, because of our belief that the diastolic sounds do not represent the closure of the semilunar valves. We might go a step farther, and say that in our belief neither represents the physiological second sound, and thus express our scepticism of theories that assume an interpolated sound to be the cause of the triple rhythm. The true *bruit de galop* is a sign always best heard over the ventricles, *i.e.* over the chief part of the muscle of the heart, and though occasionally it may be heard widely distributed and even audible at the base, in the latter situation it is less loud than over the body of the heart. The *bruit de galop* may be heard in all forms of muscle failure of the heart—even in that due to anæmia—but the most perfect examples are supplied by cases of chronic Bright's disease, when the vigour of the hypertrophied heart muscle is on the wane, and so onwards to the end. Often it is associated at one time or another or permanently with the murmur of mitral and tricuspid incompetence, but its most perfect development occurs in the absence of any murmur, and its importance as an auscultatory sign perhaps surpasses—or at least is equal to—that of any murmur, its therapeutic bearings are so significant. It *means a failing heart muscle.*

THE PULSE IN MUSCLE FAILURE OF THE HEART

The condition of the pulse in cardiac muscle failure is one of very great importance, inasmuch as it forms the guide to the method of our therapeutic endeavour. *All degrees of arterial tension may be associated with muscle failure from the highest to the lowest,* though the former cannot be expected to be long maintained in the presence of weakening of the muscle of the heart. Moreover, intermittence, inequality of beats, and all forms of irregularity up to the fancifully named "delirium cordis" pulse, are quite commonly met with. Tachycardia, too, may occur, and cannot be regarded without apprehension, seeing how it must wear out a heart when long continued. The opposite condition, bradycardia, may likewise be encountered.

It cannot be too strongly insisted on that failing heart and high arterial tension are often associated together; nay, the latter is a powerful agent in the determination of the latter. The combination is often witnessed in the course of chronic Bright's disease, and during the association it is that the most distressing dyspnœa is witnessed, a struggle, as it were, being maintained between heart and vessel.

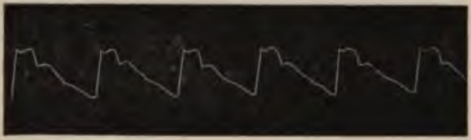

FIG. 8.—High tension pulse in case of Bright's disease associated with mitral incompetence resulting from a degree of muscle failure of the heart. Man æt. 38.

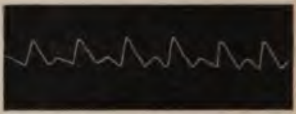

FIG. 9.—Low tension pulse. Mitral incompetence associated with endocarditis and adherent pericardium. Man æt. 25, who had rheumatic fever a year before tracing was taken.

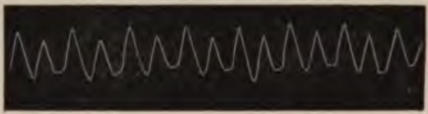

FIG. 10.—Extremely low tension pulse in septic endocarditis. Mitral incompetence associated with much damage to valves. The great size of the dicrotic wave is noteworthy, and the fact possibly bears relation to the pyrexia present.

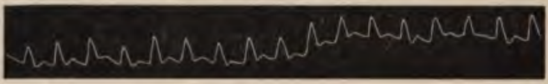

FIG. 11.—Muscle-failure in chronic Bright's disease (granular kidney, p.m.). Man æt. 47, who had at the time no incompetence of his mitral valves, and who recovered from the symptoms of cardiac failure, and lived for nearly ten years.

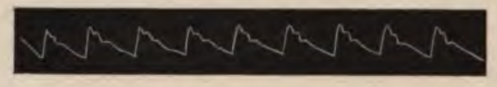

FIG. 12.—Tracing from same patient towards the end of convalescence during first stay in hospital; pulse of good, almost high tension.

As the heart muscle fails, however, the tension tends to fall, as it might naturally be expected to do under the circumstances, and it may ultimately reach a very low grade, the tidal wave of the tracing disappearing altogether, and dicrotism becoming pronounced. The presence or absence of mitral incompetence seems, however, to be in no way indicated in the sphygmogram. Irregularity is common, especially in the form of sphygmogram already described as that of the second stage pulse of mitral stenosis. But often an earlier change is occasional simple intermission. Almost as frequently there is an abortive beat represented in the sphygmogram during the intermission of the pulse that is felt at the wrist. This is the so-called "missed beat." Lately the writer met with a case which, though the intermission as felt at the wrist was only very occasional, invariably *two* in

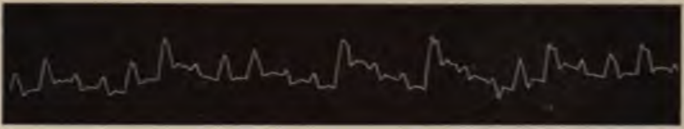

Fig. 13.—Temporary great irregularity of the pulse. "Delirium cardis." From a case of muscle failure of heart in a case of Bright's disease (gran.-kidney).

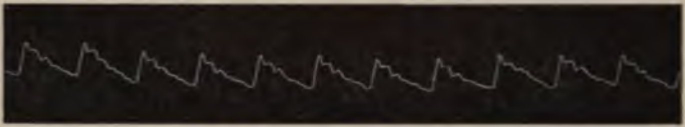

Fig. 14.—Usual pulse of same patient.

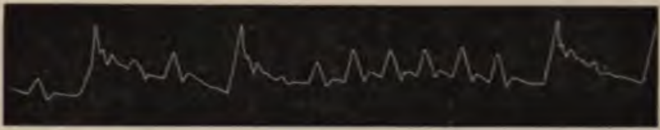

Fig. 15.—Irregular pulse in the cardiac muscle failure of Bright's disease (gran.-kidney). Patient from whom Figs. 11 and 12 were taken, but towards the end of the case. Mitral incompetence.

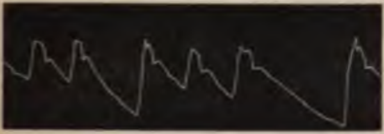

Fig. 16.—Intermission of the pulse. The long upstroke of the curve that follows the intermission is to be noted.

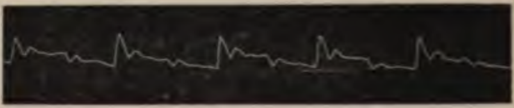

Fig. 17.—Bigeminal pulse in fatal case of alcoholic muscle failure of the heart.

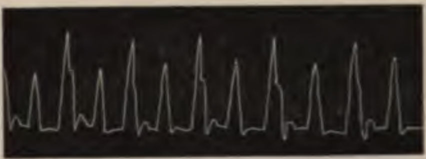

Fig. 18.—Alternating pulse in muscle failure of the heart.

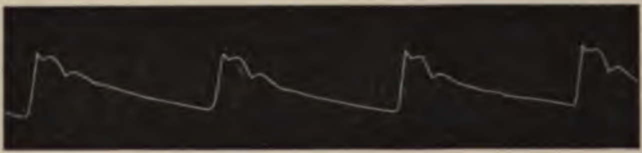

Fig. 19.—Pulse in case of typical bradycardia associated with syncopal seizures.

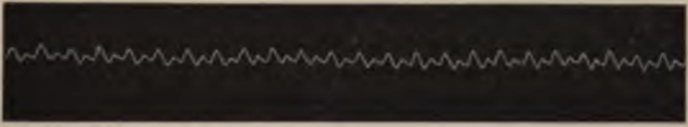

Fig. 20.—Tracing of pulse taken during an attack of tachycardia. Diabetic patient without obvious sign of cardiac disease.

place of *one* beat proved abortive—in fact there was a rare *trigeminal* beat. A continuous trigeminal pulse is rare for any length of time, but not so a bigeminal one, and though often associated with the effects of digitalis, it is by no means always so. Whenever an apparently slow pulse is felt, the possibility of there being an intermediate beat imperceptible through the finger—in other words, of our having to do with a bigeminal instead of a bradycardiac pulse—must be entertained. Examination of the heart with the stethoscope will at once indicate whether this is the case or not. The heart sounds, however, are most difficult to analyse in the presence of this peculiar action of the heart, and the writer questions the utility of entering upon a discussion of the subject. Of course murmurs are often present and add to the complexity of the combination, which may be met with in any kind of heart disease, and would seem, therefore, to be directly associated with the state of the muscle of the heart and of its nervous mechanism.

True bradycardia or slow pulse is a rather rare condition, and may be, as far as we know, congenital. It is remarkable, as we shall find, inasmuch as its effects are observed specially on the arterial side of the circulation, syncopal or epileptiform attacks being liable to result from cerebral anæmia (*vide* p. 443).

It is evident from the foregoing remarks that several quite distinct types of pulse may be met with in cases of simple muscle failure of the heart, and the variations they present may be extreme; thus there may be very high tension in the struggle against which the heart muscle is beginning to fail, and there may be extremely low tension when the heart has utterly failed or there is general vaso-motor paralysis. There may be, again, bradycardia or tachycardia and all forms of irregularity, varying from a rare intermission to a state of utter disorder in which it is impossible to recognise any prevailing type or types of curve. It follows from these remarks that from a diagnostic point of view little is to be expected from observation of the pulse in muscle failure, but it is far otherwise when therapeutic indications are sought in the same direction.

TREATMENT

The treatment of heart disease includes much more than the mere administration of drugs, powerful for good or ill though these may be. Nothing seems more rational than the enforcement of *rest* upon the cardiac sufferer. It is impossible to give his heart rest; all we can aim at is diminishing its work as far as in us lies. Of late years much has been heard of a treatment in which graduated exercise plays an essential part. This method of treatment is fully described elsewhere in this work (see Schott treatment), and it is not for the writer to express his opinion upon it, but he may be permitted to quote from Dr. Stokes' work on Heart Disease, published in 1854, the following passage to show that, as far as the exercise is concerned, the treatment is not of such recent origin as is often supposed. The patient is enjoined by Dr. Stokes "to pursue a system of graduated muscular exercise; and it will often happen that after perseverance in this system the patient will be enabled to take an amount of exercise with pleasure and advantage which at first was totally impossible owing to the difficulty of breathing which followed exertion. . . . The symptoms of debility of the heart are often removable by a regulated course of gymnastics or by pedestrian exercise, even in mountainous countries such as Switzerland or the Highlands of Scotland or Ireland." The term "debility of the heart," used by Dr. Stokes, is a singularly appropriate one, and the condition for which the treatment can be alone regarded by the writer as appropriate must be something resembling that present in the sedentary man, who, besides being often an injudicious feeder, allows his whole muscular system, including his heart, to become "flabby." This is all the reference that the writer will make to the subject, but he takes the opportunity of reminding the reader of the immense influence exerted by exercise and careful feeding in the process of athletic training, in which process no one can doubt that the heart is largely influenced—without exaggeration one may say *chiefly* influenced, seeing that it is "wind," so closely related to the heart's health, that dominates the successful result of the process. Without good "wind" dependent on a vigorous heart muscle the most perfect development of all the other muscles of the body would be rendered useless. But the fact is too obvious to require statement.

It will be generally conceded that if a man has an aneurysm of his left ventricle, the result of an obliterated coronary branch, or if one or both of the main coronary vessels are narrowed at their orifices by chronic aortitis, nothing but disaster could follow his submission to treatment by exercise. Valve defect, however, is an intensely mechanical result of disease in the first instance, and the muscle of the heart may remain for long perfectly sound in its presence, and therefore, we may say, capable of improvement in strength by the "gymnastic" process, but even in this case we must bear in mind that the heart muscle by virtue of such valve defect is always being, as it were, *exercised to the full*, and that it will not thus have the usual margin for improvement in strength. There is no valve lesion that does not throw strain upon some part of the heart muscle, and the "solidarity" of the organ must be remembered. When a patient comes seeking treatment for heart disease, it may be granted that he has, at least, the first of the cardinal symptoms—dyspnœa on exertion. Already, then, the circulation is disturbed, and the heart has begun to be overborne in the struggle. *Rest*, with the object of relieving the heart of its work, as far as that is possible, is now imperative, and *rest alone* will often suffice to turn the balance in favour of the heart, so that the circulation is restored and the disorder of functions, resulting from its disturbance, disappears. No doubt a large number of patients, who have all the three cardinal symptoms of heart disease and many subordinate ones, would recover with a profuse diuresis, unaided by any treatment by drugs, if it were the custom (and we are glad it is not) to withhold medicinal treatment for several days. The fact of some patients doing so, however, teaches an emphatic lesson as to the importance of rest, which should be absolute in order to yield its greatest benefits. Patients in advanced stages of heart disease will often say that rest in bed is impossible for them; this is true in a few cases, but only in a few. Patients, however, are often unable to lie down in bed (orthopnœa), but this is a much less important matter. When a patient will only sit in a chair, often leaning on the back of another chair or other piece of furniture, his dropsical legs are not likely to be relieved unless it be by acupuncture: on the contrary, their condition is apt to be aggravated, and the tense skin may ultimately crack and exude serum, while an erythematous condition is apt to supervene, which rarely eventuates in sloughing. It is very desirable, then, that the sufferer from heart disease should remain in bed, though it is astonishing how frequently bed is energetically resisted by the patients who require it most. In hospital practice we find, however, that the number of patients who really cannot stay in bed owing to their dyspnœa is very small indeed, apparently not so much by persuasion being more successful as by force of example. The writer has reason to believe that the imperative necessity of *rest in bed* is often not sufficiently insisted on by the private practitioner; that is to say, the patient's consent is not obtained when it easily might be. One of the dangers of heavily narcotising a patient with severe heart disease is his being suffocated by mechanical interference with the upper air-passages, by bending his neck too acutely, or letting his head press upon folded arms and so forth, and such an accident is more likely to occur when the patient sits up in a chair. In the few cases referred to in which the patient *must* sit up, this necessity will often disappear in the course of a few days, during which medicinal treatment effects amelioration. The form of dyspnœa called "Cheyne-Stokes respiration" is often associated with distressing restlessness during the dyspnœal paroxysm, the patient often actually springing out of bed during the height of the seizure, a condition that seriously militates against the chances of recovery, otherwise small enough. Active delirium, again, is a serious symptom on its own account for the same reason; but both Cheyne-Stokes respiration and delirium are symptoms for the most part met with only in the advanced stages of heart disease, and in the most unmanageable forms of the disease, as that sequential to chronic Bright's disease.

In conclusion of the subject of rest in the treatment of heart disease, the writer would emphasise the importance of a careful general survey of the whole case in determining the amount of rest to be enjoined, and especially, when that amount has been obtained, the importance of a *gradual* resumption of activity. All the benefit derivable from the rest may be flung away by injudicious management in this respect at the end. Nay, fatal syncope may be induced by the patient being *suddenly* allowed to assume his wonted activity. But short of such an accident the faith of the patient in the efficacy of his enforced rest may be so shaken that he will never again submit to rest while he has the power of resisting it.

The Weir-Mitchell treatment has always seemed to the writer a method of accomplishing rest with the great advantage of abundant nourishment at the same time being rendered innocuous in spite of the denial of voluntary activity to the muscles. The muscles are "exercised," as it were, by the masseur—that is to say, are helped to the disposal and removal of their often-toxic products of metabolism—without any strain on the heart. Rather, we might fancy, does the process actually relieve the heart by rendering the peripheral circulation more easy. Of course, in the presence of much general dropsy, the kneading of the muscles is rendered impossible; but it is not under these circumstances that the question of this treatment is likely to arise.

DIET

We have next to consider that most important subject in the treatment of heart disease—diet. That this subject is too often ignored by the medical practitioner is abundantly evident by the frequent bitter complaint by the patient of his gaseously distended abdomen, which, pressing up on his diaphragm and impeding his already embarrassed respiration, occasions him infinite discomfort. If there is some albumin in his urine, as is the rule in most severe cases, so much the more is the patient likely to be injuriously fed with excess of carbohydrates, rice, tapioca, and the like pernicious puddings, while his nitrogenous food is cut down to a minimum. If we reflect that the heart is a muscle, and the most important muscle—not to say the most important organ—in the body, surely we ought to give the food that long experience has taught us is best for muscle tissue—nitrogenous, and not carbohydrate articles. The modern system of athletic training is, the writer understands, less strict in this respect than that of former days, but he doubts if the "staying powers" of its subjects are any better in consequence—if as good. However, past records are good enough for him. The patient with severe heart disease cannot take exercise, and we must recognise the fact. His muscles must lie fallow, except in the very few cases in which general massage is adopted. But is the case for excess of carbohydrate food any better on that account than that for a preponderance of nitrogenous food? If the physician fears an excessively nitrogenous diet, let him reflect that the toxic nitrogenous bodies in the blood he fears are promoted, not diminished, by a largely carbohydrate diet, inasmuch as the articles of the latter will use up the oxygen necessary for the metabolism of the tissues and for dealing with its nitrogenous products. Another consideration is the state of the liver, in all cases with venous stasis: the nitrogenous elements of food and the nitrogenous products of metabolism no doubt undergo change in the organ; but so also—and probably in greater amount—do the carbohydrates, so that the abnormal state of the gland that must result from its congestion with venous blood is at least as important in the one case as in the other. We should say that excess of carbohydrates in the diet was infinitely worse than an excess of nitrogenous elements, provided that carbohydrates are at the same time cut down to a minimum. So much for the question of metabolism. But for the primary disposal of food in the stomach what does reflection teach? A rice pudding alone is probably rapidly passed through the stomach, and does no mischief there; but far otherwise is the case, if nitrogenous food, as beef or mutton, is given at the same time. Evidently the rice pudding will simply clog the process of gastric digestion by its bulk, and if digestion is weak it is only too apt to be arrested altogether, a decomposing mass of incongruous stuff being ultimately passed into the intestine to create discomfort lower down. We may start with the assumption, well borne out by the scientific labours of the late Sir William Roberts, that the healthy human being has great excess of digestive power, and can afford to conform to the customs of society in the mixing of his foods. Most healthy people can do this up to a good old age; nevertheless, in many difficulty of gastric digestion is experienced early in middle life, in which case relief can often be obtained without medicinal agency by the mere simplification of the dietary; that is to say, the patient lets his stomach's full energy be exerted on his nitrogenous food, while he is equally careful to take his carbohydrate food at a different time from that at which he takes his nitrogenous food, thereby, no doubt, promoting its speedy passage through the stomach to the parts beyond, which are capable of dealing with it, all the more that the saliva, with which it ought to be saturated, has probably to a large extent escaped being interfered with by the acid of the juice in its rapid transit through the stomach. We plead, then, in the case of cardiac sufferers, for *simplification of their food*, in the first instance, and in the second for the *reduction to a minimum of their carbohydrate food;* the object of the first principle concerning the primary digestion in the stomach, that of the second principle concerning the metabolism of the tissues. We have spoken only of the stomach, but the digestion of food and the impeded absorption of food and fluid throughout the whole tract of the alimentary canal beyond must not be forgotten in a heart case. We have dwelt in symptomatology on the distressing tympanites of heart sufferers, and in its production the important part played by food is only too patent. Far better than any drug treatment, by intestinal antiseptics, is the simplification of the alimentation of the sufferer, bearing in mind his peculiar disabilities and how all his digestive powers are handicapped by the venous stasis of his digestive organs, which, judging from the condition of the spleen, would be still worse were it not for the ready distensibility of the liver.

Let us next inquire how these principles may be carried into effect in actual practice. The sufferer from heart disease is usually free from fever, and there is no necessity, unless he has temporary gastric catarrh, to feed him on "slops"; there is no reason, usually, why he should not retain the ordinary habits of health and have three meals a day. These should be small in bulk, easy of digestion, and arranged at sufficient intervals to permit of complete removal of one meal from the stomach before the entrance of the next into it, and of course a period of inactivity should be allowed to the organ. Long fasting, as from breakfast to late dinner, is to be deprecated. This habit is not infrequent in the earlier stages of heart disease, while the patient is still able to attend to his business. The meals should, further, be as *dry* as possible, with the exception of breakfast, and on this account only a small quantity of soup (and that clear) is permissible — but soups are not to be recommended. Half a glass of whisky or brandy in half a small tumbler of water may be allowed with the mid-day and evening meals. Breakfast should form the carbohydrate meal of the day, and may consist of thin crisp toast "done through," and not having a spongy centre, which is to be buttered cold, care being taken to have the butter of the best quality, and of a soft-boiled egg, or even two. A large cup of boiled milk, flavoured with coffee or Chinese tea, and sweetened with saxin, should constitute the fluid of the meal. If the patient craves for it, and it is found to agree, the *fat* of bacon may be allowed. The egg may be poached if preferred, or even fried. If the patient cares for it, fruit may be allowed. Eight or nine o'clock will usually be the time chosen for breakfast, and the mid-day meal will then be taken at 1 or 2 P.M. Cups of bovril and the like are to be deprecated in the forenoon. The mid-day meal should consist of a chop, fish, or fowl, with some well-cooked *green* vegetables—cabbage, spinach, and the like. Boiled cabbage may be squeezed through a sieve so as to break up its fibres, and should be cooked with plenty of butter, if this does not disagree. Reference to butter reminds the writer to allude to the not uncommon experience that certain patients are intolerant of fats, which it is difficult to get them to take, while others, who often experience a difficulty in the digestion of starchy foods, have a great capacity for consuming fats. These and other idiosyncrasies, such as inability to eat the smallest particle of an egg without suffering, must be recognised and respected when they are ascertained to be well founded. With the mid-day meal fruit in season, either cooked (saxin being added if sweetening is necessary) or in the natural state, may be allowed. Cheese is apt to prove indigestible; but some patients have a surprising facility in digesting it, and to them a small amount may be allowed along with butter, but on no account is bread or biscuit to be taken along with it. At five o'clock a cup of pure Chinese tea, sweetened with saxin, may be taken, *but no bread or biscuit on any account.* At 7 or 7.30 P.M. the evening meal is taken, and should be, in principle, practically a repetition of the mid-day meal; only, if chop has formed the latter, fish, or fowl, or a joint may constitute the former, and so on. Green vegetables are to be taken again. Patients often demur to taking green vegetables, under the impression that these increase tympanites. This is not the writer's experience when carbohydrates are at the same time withheld. There is no great objection to a double course—say, fish and a joint,—or occasionally a *small* quantity of clear soup may precede the joint, fish, or fowl, as it happens to be. Fruit may follow; cheese is perhaps better avoided. These three small meals will usually be abundance for the cardiac sufferer; more he probably could not utilise, even though he were capable of properly digesting it. The writer used to feed his patients more largely than experience has since taught him to be desirable.

A glass of whisky or brandy in half a tumbler of hot water about eleven or twelve o'clock will often help the patient to a good night, but *on no account must any biscuit or bread be taken along with it.* In the case of the so-called working classes the writer is continually contending against the terrible "tea-meal" that patients of this class are so reluctant to give up. In a class somewhat higher, this same meal is made still more noxious by some nitrogenous elements of food being thrown in—fish, chops, and the like—to form in the stomach, amid a large amount of bread and butter, and tea and jam, a mass indeed well calculated to frustrate the efforts of the most potent juice to act upon it. The writer has long abandoned the names "dinner" and "supper" for the mid-day and evening meals, which are simply so termed; he is convinced that our working classes would live healthier lives if they deferred the principal meal of the day till evening—say, till an hour after their return from work, which hour is well spent in repose. He has been told that the principal meal is requisite in the middle of the day in order to supply strength and energy for the labour of the day. The taking of a *small nutritious* meal in the middle of the day, and the reservation of the principal meal till the day's work is over, would in his opinion be a much better system, a substantial breakfast being of course requisite.

As already stated, it is astonishing how well, in many heart cases, the digestive powers are retained even in the presence of considerable portal congestion, nay almost to the end of the case. But portal congestion unquestionably implies a proneness to catarrhal inflammation of both stomach and bowels. It is in the case of the former organ that this process is most evident—the patient losing all appetite and then vomiting his food. (The possibility of digitalis being a factor in the sickness must always be borne in mind.) For this condition medicinal treatment alone avails little. Large doses of subnitrate of bismuth constitute probably the best form of it, but in a severe attack deprivation of all food by the mouth is necessary, nutritive suppositories or enemata being given per rectum. In any case the food taken by the mouth should be limited to milk, and this is best peptonised, as there can be very little digestive power in the stomach during catarrh. Fermented milk or "koumiss" (originally mare's milk) will often be retained when ordinary milk is rejected. But it fails in a certain proportion of cases, and then there is no alternative but to let the stomach have absolute rest for a time; usually twenty-four or forty-eight hours suffice for recovery. Care, of course, must be exercised in the resumption of nourishment by the stomach—only bland fluids in small quantities at a time are at first permissible.

Mention must be made of an absolute milk diet in the treatment of heart cases, especially those associated with high arterial tension and kidney disease. This treatment is only applicable to the patient confined to bed. It would seem that the products of metabolism arising during milk diet and rest are much less toxic than those produced while ordinary diet and exercise are being taken; and as a consequence the vascular tension declines, an important matter, seeing that it directly concerns the heart. No doubt another element in the good effects of the treatment is the speedy washing away of any toxic products of metabolism there may be. A tendency to constipation can easily be obviated by laxative drugs. But few patients will or can continue the treatment long.

It is such a common practice to give frequent purgatives in heart disease, that it is usually hard to disassociate catarrh of the bowels from their action. Moreover, the mucous membrane of the intestines in a severe heart case is sure to be exposed to the venous stasis of the portal system. Notwithstanding, it is not often that diarrhœa calls for treatment, a moderate looseness being usually considered desirable. It must be remembered, however, that any approach to acuteness of the catarrhal process is apt to exert a depressing influence upon the heart; while frequent actions of the bowels imply exertion on the part of patients who do not use a bed-pan. These considerations lend additional importance to the dietetics of heart disease, and the writer believes that tympanites, so distressing to many sufferers from the disease, is largely promoted by excess of carbohydrates—the usually innocently regarded rice pudding and its kindred—while well-boiled green vegetables and even fruit may be taken with impunity provided they are taken with nitrogenous food alone. It is a remarkable fact that cases of obstinate chronic tropical dysentery after resisting all medicinal treatment are sometimes cured expeditiously and permanently by the consumption of large quantities of fruit, apples, etc. The permission to take fruit may seem to be contrary to the principles of diet already laid down, but a patient, whose starchy food and cane-sugar is as much reduced as has been advised in heart cases, can consume the glucose of fruit with as much impunity as the patient on milk diet consumes his lactose. We are not dealing with diabetic patients, though an apparent sugar reaction is sometimes obtained in the urine of cardiac patients; generally it is due to excess of uric acid, or the presence of some abnormal body, as glycuronic acid; sometimes really to glucose. When we consider the disorder of the liver that must result from heart disease, it is surprising rather that glycosuria should be so rare than that it should occur occasionally in the urine.

Medicinal Treatment.—We have next to consider in detail the treatment of heart disease by drugs. The powerful and toxic remedies that act directly on the heart muscle itself must of necessity have the first place in the consideration of these remedies. One may be said to be *facile princeps* —a giant among the rest—namely, digitalis, which, take it all and all, has no rival. A knowledge of the peculiarities of action of this remedy, which include the slowness of the establishment of its diuretic effects, its cumulative properties, by virtue of which its action is maintained for a considerable time after its administration has ceased, and its double, in a sense antagonistic action, at the same time on both the heart muscle and the peripheral arterioles, is of immense importance in the employment of digitalis in practice. The effect desired is a tonic one on the heart muscle, the best indication of the achievement of which is an increase in the flow of urine steadily maintained for several days. And once for all, let it be clearly understood that it is the *heart muscle* and not any *lesion* there may be, that is influenced by the drug. The writer has absolutely no faith in the suitableness of the remedy in cases of one lesion, and in its unsuitableness in cases of another lesion. Such views, he thinks, have sadly often led to the withholding of digitalis, when it constituted the only hope of the redemption of the case. Aortic incompetence and mitral stenosis are the two lesions in regard to which purely theoretical considerations have been allowed to exert such a grievous influence on practice.

With regard to the former, the writer can only endorse the view of his old master, Dr. G. W. Balfour, and maintain that aortic incompetence, far from affording cases that are unsuitable for the administration of digitalis, supplies the most brilliant examples of its curative effects. He would go further, and add that on calm reflection theory itself bears out the actual clinical experience of the treatment of cases of the kind by digitalis. Aortic incompetence is the lesion of all others whose effects fall directly upon the left ventricle, the most powerful part of the heart muscle, and when we give digitalis under the circumstances we get its effects exerted in chief force just where it is most wanted. In mitral stenosis, a better case for the view he has condemned the writer admits can be made out; for in its case we have no powerful muscle wall in the chamber first concerned to influence and stimulate. Before we can bring increased muscle vigour to bear on the obstruction the pulmonary circulation must be congested, and after all, the right ventricle is at best wretchedly weak in comparison with the left ventricle. Moreover, in the earlier stages of the lesion the pulse, as we have found, is one of considerable tension, the sphygmogram displaying a well-developed tidal wave. But practical experience overrides these considerations. The study of cases of mitral stenosis to their end brings fresh strength to the belief in the practical usefulness of digitalis, for in the vast majority of cases the *left ventricle is enlarged*. When this unquestionable fact is explained in as unquestionable a manner, the writer is prepared to reconsider his position in regard to the efficacy or harmfulness (for if it does not do good, so powerful a remedy cannot be simply inoperative) of digitalis in mitral stenosis. The writer cannot too strongly condemn, however, the custom of giving digitalis as soon as the lesion of mitral stenosis is discovered, and while there are practically no symptoms, and there is a regular and almost too high pulse tension. But, of course, in every form of heart disease it is the evidence of *muscle failure* that alone constitutes the justification of the use of digitalis and the other drugs of its class. The pernicious habit of giving such drugs as soon as a lesion is discovered is to be further condemned, in that it probably, so to speak, "spoils the case," by habituating the heart muscle to its influence, in such a way that when the time comes for actual muscle failure and for the physiological effects of such drugs being legitimately sought, the heart muscle has been so rendered abnormal with regard to them that only an abnormal response can be forthcoming. These remarks are meant, of course, to apply chiefly to digitalis, but the same meddlesomeness may make use of other tools.

In certain of the lower animals death from digitalis poisoning is associated with tetanic contraction of the heart, which of course brings the circulation to a stand-still quite as effectually as the most complete paralysis, but in the human subject all post-mortem evidence goes to show that the ventricles are paralysed and not tetanic when death occurs under the influence of digitalis. Nevertheless the fact need not shake our faith in digitalis as a muscle stimulant, inasmuch as with most active drugs the excessive action is the opposite in nature from the feeble and moderate, and "stimulation followed by paralysis" is an oft-told tale in descriptions of the effects of poisons.

When we overdo the effects of digitalis it is paralysis and not spasm of the heart that we must apprehend, but, most fortunately, in one sense digitalis is the safest of poisons inasmuch as it gives to the *observant* prolonged and emphatic warning when it is being pushed too far. This, however, is absolutely true of cases in which rest in bed is insisted upon. When digitalis is given to patients "going about," as the phrase is, it must be given in exceedingly small dose and—what is more important even—in *short courses*. That is to say, the drug must be intermitted for several days between each course, for its effects take a considerable time to pass off. There are few drugs, however, that in their administration require so much carefulness on the part of the practitioner as digitalis. It is necessary, therefore, that we should consider the mode of application of the remedy in some detail, for misused it becomes a dangerous poison—

peculiarly dangerous in the fact that the symptoms of poisoning may readily be *overlooked until the sudden lethal termination of the case,* and in that the patient may be *poisoned without ever having taken a single dose beyond that rightly laid down by authority as safe.* The drug is "cumulative," as it is termed; its toxic effects are developed by *prolongation* of administration in *small and infrequent doses,* without the intervention of a pause sufficiently long to permit of the elimination of its influence. In many cases, fortunately, vomiting and abdominal pain give warning of over-action, while the pulse usually becomes slow and often bigeminal or irregular. But the careful practitioner will seldom let the administration proceed so far as the production of this condition, for he will anticipate and obviate its occurrence. All the official preparations of digitalis are reliable enough, and it is hard to choose between them. The tincture is probably most frequently used, but the infusion seems to be the most active preparation. The powdered leaves are admirable for a method of administration, similar to that to be immediately recommended with regard to a French preparation that the writer has used for many years almost to the exclusion of the official preparations. He has, however, employed this preparation simply for the sake of convenience, and not because he believes that it possesses any inherent virtue absent in the ordinary preparations. Of the activities of the crude drug it may be said to possess both the best and the worst—the most potent and most cumulative. This preparation is the granule of M. Nativelle of Paris, containing $\frac{1}{240}$ of a grain (or $\frac{1}{4}$ of a milligramme) of "chemically pure crystallised digitaline." Into the question of the precise nature of this active principle the writer will not enter, but with regard to its activity and its equal distribution in the granules he has had large experience. M. Nativelle makes also a syrup of digitaline, each teaspoonful of which is equivalent to a granule, and contains $\frac{1}{240}$ grain of "crystallised digitaline." This is a convenient preparation for use, when a smaller dose than ordinary is required, as in the case of children.

When the writer first began using the granules of Nativelle, he believed the statement that one granule was equivalent to ten minims of tincture of digitalis, B.P., and therefore that a granule may be administered precisely in the same way as he then was in the habit of administering the tincture, that is to say, three times a "day," equivalent to three doses in twelve hours instead of twenty-four hours, or, one may say, every four hours during the day proper, and none at all during the "night," or remaining twelve hours. The result was almost invariably toxic symptoms, so that for a considerable time he gave up the use of the granules altogether. Evidently the granule consisting of one ingredient of the crude drug and the official preparations of the latter should be regarded as incomparable, and even of the latter it is a faulty mode of administration to give three doses in the first twelve hours and none at all in the second twelve hours. When it is desired to "push" quickly the action of digitalis, three granules may be given in twenty-four hours, but each must be given after an interval of *eight*—not four—hours. Usually, however, one granule in twelve hours is amply sufficient—two a day. Even this number can seldom be continued long with advantage, and usually in a very few days one granule in twenty-four hours suffices. In a few days more the drug may be stopped altogether, or intermitted for forty-eight or seventy-two hours according to circumstances. The dose must never be increased after the administration has continued for some time, a rule the necessity of which is obvious from the cumulative property of the remedy, and the dose must always be diminished as the course proceeds for the same reason.

The quantity of urine passed by the patient forms by far the best guide to the effects of digitalis, and with reference to it we must bear in mind that digitalis is a slow diuretic, requiring at least forty-eight or seventy-two hours for the production of polyuria. Thus if a patient, on the day after the one on which digitalis was first administered, gets a profuse diuresis, such diuresis cannot be attributed to the drug; all experience is opposed to such a diuresis being the effect of the drug, in all probability it is "spontaneous" or the effect of rest. But if the diuretic effect of digitalis is never obtained promptly, it is occasionally delayed for a considerable number of days. A curious experience is the following: a patient has been taking digitalis, say for ten days, while there has been no material increase of the urine-flow, and the practitioner, despairing of success, stops the remedy, and orders a drug without diuretic property, say liq. strychniæ, when, to his surprise, within seventy-two hours after the last dose of digitalis a profuse diuresis sets in. Such an occurrence is by no means rare, and indicates that the administration of the drug has been pushed too far, its effects being possibly exerted specially on the arterioles, which become too much contracted in the kidneys to permit of a diuresis which is rendered possible only when the vessels have relaxed to some extent under the diminishing influence of the drug. The possible advantage of combining a vaso-dilator with digitalis will be referred to later. When a diuresis is thoroughly established it is well to diminish the dose of digitalis, or to give the same dose less frequently, seeing that its effects thereby will be efficiently maintained, while the risk of them being over-exerted is obviated. The most difficult question is, what to do with regard to continuing the administration in cases in which there has been no response by diuresis at all. If in doubt, the rule should be to intermit the drug for at least a couple of days and probably longer. The desirableness of resuming the drug or replacing it will then have to be considered afresh.

Strophanthus and other Cardiac Remedies

The drug that undoubtedly ranks next after digitalis in the treatment of heart disease is strophanthus, usually prescribed in the form of tincture. From experimental results it would seem to have an advantage over digitalis in some respects, exerting less influence on the peripheral circulation in the direction of contraction of the arterioles. It is not quite clear, however, that this is an unqualified gain; moreover, we have found that the results of the experimentalist in his laboratory and of the physician in his ward are not entirely in accord with regard to such a decisive question as that having reference to the ultimate effects of digitalis upon the heart: in fact as to whether the heart under the full influence of this drug is found after death in systole or diastole. Of one thing the writer is convinced from his own clinical observations, and it is this: that in the case of patients who are admitted to hospital with low arterial tension, and who ultimately recover a high degree of arterial tension, the diuresis that is the indication of the successful administration of digitalis precedes in the great majority of cases the recovery of arterial tension.

Strophanthus seems to be certainly a less cumulative drug than digitalis, but, if its administration be long continued, similar toxic symptoms, including abdominal pain, vomiting, and irregular action of the heart, are apt to be developed. The writer possesses charts to show that sometimes strophanthus will succeed after digitalis has apparently failed to produce a diuresis, and *vice versa*. In the former case, however, it is at least open to question if the influence of the digitalis has not had a share in the result. He has not found strophanthus useful in irregular action of the heart *per se;* he has thought, indeed, that it at times aggravated the irregularity. He has, moreover, been quite unable to formulate rules for guidance in the preference of strophanthus and digitalis, and he recalls one case of grave damage to the mitral valves and much dilatation of the heart in a rheumatic girl, in which digitalis always failed to benefit in the slightest degree, while strophanthus as regularly was followed by the best results, the experiment being repeated on several—many—occasions during the long period of the patient's residence in hospital. It may be added that the pulse tension was very low, altogether in opposition to theoretical considerations concerning indications for the preference of strophanthus.

There are numerous drugs capable of producing diuresis in heart disease, which are seldom given a fair chance, seeing that they are only used after digitalis at least has failed, and often after both digitalis and strophanthus have done so: convallaria, apocynum, scoparius, etc.

Then there is the series of drugs that seem to act on the kidney, rather than the heart in the production of diuresis—diuretin (Knoll) is probably the best example of this class. The writer recalls an exceedingly grave case of aortic incompetence which seemed steadily going from bad to worse in spite of digitalis, strophanthus, etc., but in which there was the remarkable (under the circumstances) fact of the absence of even a trace of albumin from the urine. Basing his action on the soundness of the kidneys, he ordered diuretin—2 grs. every hour—with the result of establishing a profuse and long-maintained diuresis, which removed all dropsy and enabled the patient, to the marvel of physicians and nurses alike, to leave the hospital for his own home, much relieved. In cases neither cardiac nor renal, again, he has found the drug useful in setting up a diuresis for the purpose of removing local dropsies of inflammatory origin, as pleuritic effusion. Sometimes the drug produces sickness, which prevents the continuance of its use. It is the diuretic of all others, whose effects the writer has been able repeatedly to reproduce at intervals. It is probable that diuretin acts specially by stimulating the kidney, and the writer believes that he has seen it twice produce hæmaturia in cases of tubular nephritis, as if by over-stimulation. Citrate of caffeine, to which diuretin is closely related, is another drug that acts principally on the kidney; most admirable results are sometimes obtained by its use, but it is somewhat capricious—or perhaps seemingly capricious—in its effects, of which we have not yet a perfect knowledge.

In considering the records of the past concerning the treatment of heart disease, one cannot fail to be struck by the high place among remedies given by the old physicians to purgatives. That these were often used to the detriment of the patient cannot be doubted, but it is no less certain that our forefathers, who were keen observers, based their practice on a measure of experience. At the present time it is *discrimination* in the use of purgatives that is the desideratum. Such discrimination must be based chiefly upon the condition of the pulse, or, to put the matter more precisely, upon the arterial tension. It cannot be too strongly insisted upon, that the finger must not be trusted to indicate the degree of vascular tension with absolute precision, while a sphygmogram can be taken easily within five minutes even in difficult cases, and at once indicates the nature of the pulse with which we have to deal. It is necessary here again to refer to the fact, already previously emphasised, that a high degree of tension is not rarely—not possibly—but is *commonly* associated with a labouring failing heart. No doubt, provided the cardiac failure be progressive, so will be progressive the lowering of arterial tension, but the process may be a long one, and of immense importance is the fact that the patient may not survive it.

That a sharp purgative by its action tends to reduce arterial tension is certain, and we cannot be surprised that it is so, in view of the tremendous influence exerted by the state of the vaso-motor nervous mechanism of the abdominal vascular system upon the distribution of the blood as a whole, as we see that influence exerted after surgical shock. The mere contemplation of such an influence may well give us pause, when we think of ordering a sharp purge in an advanced cardiac case with the pulse flickering at the wrist. Few medical practitioners of large experience can look back over many years without recalling the disastrous effects of a purgative given when the heart was very weak, and it need not be a "diseased heart" in the ordinary acceptation of the term. Even the use of an enema under like circumstances is well known to be rarely followed by disastrous—even lethal—consequences. These remarks must suffice to warn the reader against the indiscriminate use of sharp purgatives. On the other hand, in cases of high arterial tension with labouring heart, they bring much aid to the overburdened organ by lightening its load, and no doubt help to clear the blood of injurious toxins.

Space will not permit our considering the *classes* of purgatives *seriatim*, and we will refer only to special drugs and compounds. Few purgatives can rival the pulv. Jalapæ co. of the B.P., which, however, at least in hospital practice, has to be given in larger doses than the official—60 grains being not at all too much in many cases. Sir William Broadbent believes that mercury has special virtue in lowering arterial tension, and in this relation its well-known antiseptic powers in relation to toxins in the alimentary canal is worthy of note. On theoretical grounds the saline purgatives may be thought to be specially applicable in the treatment of heart disease, as the free exudation of fluid they occasion into the intestine must relieve the congestion of the portal circulation and of the liver. For this purpose they ought not to be administered largely diluted. But when given alone they do not stimulate the peristaltic movements of the bowel sufficiently to ensure the quick removal of the exuded fluid, so that it is actually possible that this may be later reabsorbed into the vessels. It is desirable, therefore, to combine the salines, of which sulphate of magnesium is the one most commonly used in the treatment of heart disease, with some drug capable of stimulating the bowel to increased peristalsis. The "black draught" of most hospitals is some such combination, to which a carminative is added. One sharp purge is better than the frequent repetition of less active ones; we hold that patients with heart disease should not be exhausted and their hearts thus still further debilitated by frequent or continuous purgation. The great indication for purgation is high arterial tension, and the many forms of cardiac failure so associated no doubt formed the ground on which the common practice of purgation in heart disease was based, that is to say, it is in cases of this kind that the best results are obtained from purgatives.

There is one remedy that so strongly makes for righteousness in treatment that it cannot possibly be ignored in an account of the management of cardiac cases:—It is morphine administered hypodermically, and usually guarded by a minute dose of atropine. Nevertheless, its use is directed to the relief of one distressing symptom only of cardiac failure, and not to the restoration of the circulation. The symptom in question is *dyspnœa*—the symptom which surpasses even angina pectoris as a cause of suffering when we regard its frequency and duration in comparison with those of angina. To Dr. Clifford Allbutt we are indebted for the introduction of this precious remedy. It should always be given hypodermically, and, as a rule, combined with atropine. It is impossible to lay down rules in detail as to dose. The treatment should be begun with infinitesimally small doses in order to see how the patient bears it, and then it is easy to increase the dose up to the point of affording relief without incurring risk. The frequency of albuminuria in cardiac cases without serious implication of the kidneys must be borne in mind. Moreover, in the distressing dyspnœa of the cardiac failure of actual Bright's disease the writer has repeatedly found the remedy to act like a charm, and to prolong life in comparative comfort, only in cases of the kind an exceedingly small dose, say $\frac{1}{20}$ of a grain well guarded with atropine, must be used at first, though it will often be found that $\frac{1}{4}$ of a grain of morphine can be given with perfect safety and with perfect effectiveness. Blocking of the bronchial tubes with secretion the writer regards as a stronger contra-indication as to the use of morphine than kidney complication. In heart cases which require no remedies directed immediately to the heart, but in which it is all-important to maintain the general health at a high standard, a mixture of liquor strychniæ and liq. arsenici hydrochlor. 4 or 5 minims of each, given in water simply and after meals, will be found very useful. Occasionally it is well to stop the arsenic, and give the strychnia alone, and before instead of after meals. Iron, no doubt, is an excellent general tonic, and a precious remedy when there is anæmia in a heart case; but the writer is inclined to think it has become too much a matter of routine to give it to patients suffering from heart disease, and in most of those in middle life and older, whose cardiac failure is apt to be associated with high arterial tension, he thinks it often does harm rather than good.

Sleeplessness is not rarely a distressing symptom in heart disease, and one which in it, as in pneumonia and typhus, taxes the resource of the physician to the utmost. Timidity and rashness must be equally eschewed. Those who err on the side of the former the writer would remind that sleeplessness is a terrible "tendency to death" in all these diseases, and that to let a patient die without attempting to save him is as bad as running risk in the attempt. But happily, with ordinary care, such risk is infinitesimal, and lies specially in lung and kidney complications. In heart cases insomnia is often associated with dyspnœa, and the remedy which is most efficient for the latter is best for the former, namely, morphia, given hypodermically. Among other drugs that may be used for sleeplessness are chloralamide and urethane. The approximate dose of the latter—which must be determined by the circumstances of the case—should be kept dissolved in sp. vin. rectif., and added to water at the time of administration. With regard to urethane, it is a safe but a weak drug, and has generally to be given in large doses. Shortly after the drug was introduced, a patient of the writer's, a bad rheumatic heart case, received by accident an overdose, but the only undue consequence was that the patient, as well as sleeping during the night, slept nearly all the following day, respiration and circulation being well maintained, and only benefit resulting.

There are very few heart cases, other than those associated with aortic disease in the wide sense, that depend on muscle failure promoted by or actually dependent on syphilitic lesions of the myocardium,[1] and the possibility of antisyphilitic therapeutics being beneficial, if not curative, of the case must not be lost sight of. The patient will probably have indications of heart failure, the common antecedents of which are absent while he has had syphilis. It must be remembered, however, that because a lesion is syphilitic it is not therefore removable by mercury and iodide of potassium; the tissue destroyed cannot be restored, although the neoplasm may be atrophied.

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[1] *See also* "Syphilis."

A drug that has come greatly into vogue of late is liquor strychniæ (or an equivalent preparation) in the treatment of advanced heart disease. The writer regards it as essentially a respiratory stimulant, and of its value as such in these cardiac cases there can be no question. It is best given hypodermically, but this method is naturally not to be recommended until the case has become very serious. Atropine has a somewhat similar stimulant action on the respiratory centre, but its use is almost confined in the treatment of heart disease to guarding, as it were, the use of morphine. Strychnia may be used for the same purpose.

The use of strychnia in early stages of heart disease as a general tonic has been already referred to.

The consideration of the treatment of the special ailments that result from the disturbed circulation of the various organs in heart disease may be carried to indefinite length, but too much attention to these is apt to throw into the background the great and essential "tendency to death," namely, *heart failure*, of which these ailments are for the most part comparatively insignificant consequences. If we succeed in restoring the heart, all such ailments will speedily be relieved. On the other hand, the most efficient treatment directed to local consequences in the different organs must fail, if no improvement in the general circulation is accomplished, but this statement must not be taken to imply that local symptoms are not to receive local treatment according to the dictates of common sense and common medical knowledge.

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Heart, Neuroses of.

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See also Angina Pectoris.

By a neurosis is usually meant a disorder, the manifestations of which are attributed to an influence of the nervous system, the nature of which is quite or largely unknown. Under these circumstances, signs and symptoms are apt to be raised to the rank of diseases, and words made to take the place of things.

Such a state of matters tends, naturally, to unreasoning or empiric treatment, which in the case of some so-called neuroses of the heart may be neither successful nor safe. These are divisible into two great classes representative of the two chief properties of the nervous system—sensibility and motility. They are classed as disorders of cardiac sensibility and cardiac motion respectively, and may, to all appearance, in some cases bear the relation one to another of cause and effect, as when the pain of angina is followed by a rescuing acceleration of the heart's action.

In the preface to the section dealing with diseases of the heart (p. 325) it was pointed out that the essential factors in the functional unity of the heart's action were the muscle cell, its blood-supply, and its nervous endowment. In examining the nature and determining the preponderant character of cases when classifying cardiac neuroses, as well as in prescribing treatment, these factors have constantly to be borne in mind.

For information regarding sensory disorders of the heart the reader is referred to the section on Angina Pectoris, and its discrimination from non-cardiac dysæsthesiæ in the region of the heart (vol. i. p. 347). In this place we shall be concerned with the nature and treatment of motor neuroses of the heart.

In the introductory chapter it was pointed out that a rhythmical pulsation of the embryonic heart was to be detected at a stage prior to the organisation of the blood-vessels and the intrusion of the nerves. Life is manifested in the cardiac muscle, in other words, before the complete formation of the vessels into which the heart is destined to project blood, and before the nerves destined to regulate its action have grown into it and been distributed to its essential cells. In course of time, however, it was also pointed out that such a distribution does take place, and is not without an influence upon its movements. Regulation of rhythmicality appears to be as necessary to its persistence as nutrition of the structures manifesting movement. The existence of rhythmical structures not endowed with nerves may now be denied as positively as the existence of such structures unnourished by blood. It is evident, therefore, that irregularities and abnormal pauses in the action of structures which are perfectly rhythmical under normal circumstances must in some measure be due, either to the abnormal exercise of the regulative influence, or to the exercise of this influence being abnormally in abeyance or impaired. It was likewise argued that the structure common to the anabolic and catabolic nerves was the muscle cell—that it was, in short, the peripheral conducting medium between the two adjuvant and supplementary if not antagonistic series of nerves. The muscular or other peripheral organic cell closes the neuro-cellular circuit at one end, just as the cells of the cortical and subcortical centres of reflex action, physical and psychical, do at the other. Continued defect, therefore, in any one of these three factors which constitute the functional unity of cardiac, as indeed of all other visceral action, may lead to disorder of the regular action normal to the organ (see Plate).

Regarding the second sound of the heart as a passive event, and the double contraction of the auricle and ventricle as one in propulsive effect, the normal action of the heart may be viewed as consisting of a regular alternation of action and pause, of work and rest, distributed in due and recognised proportions, the consequence of which, *ceteris paribus*, is a recognised normal *rate* of action. The latter, naturally, varies with circumstances. The heart of the human fœtus, notwithstanding its

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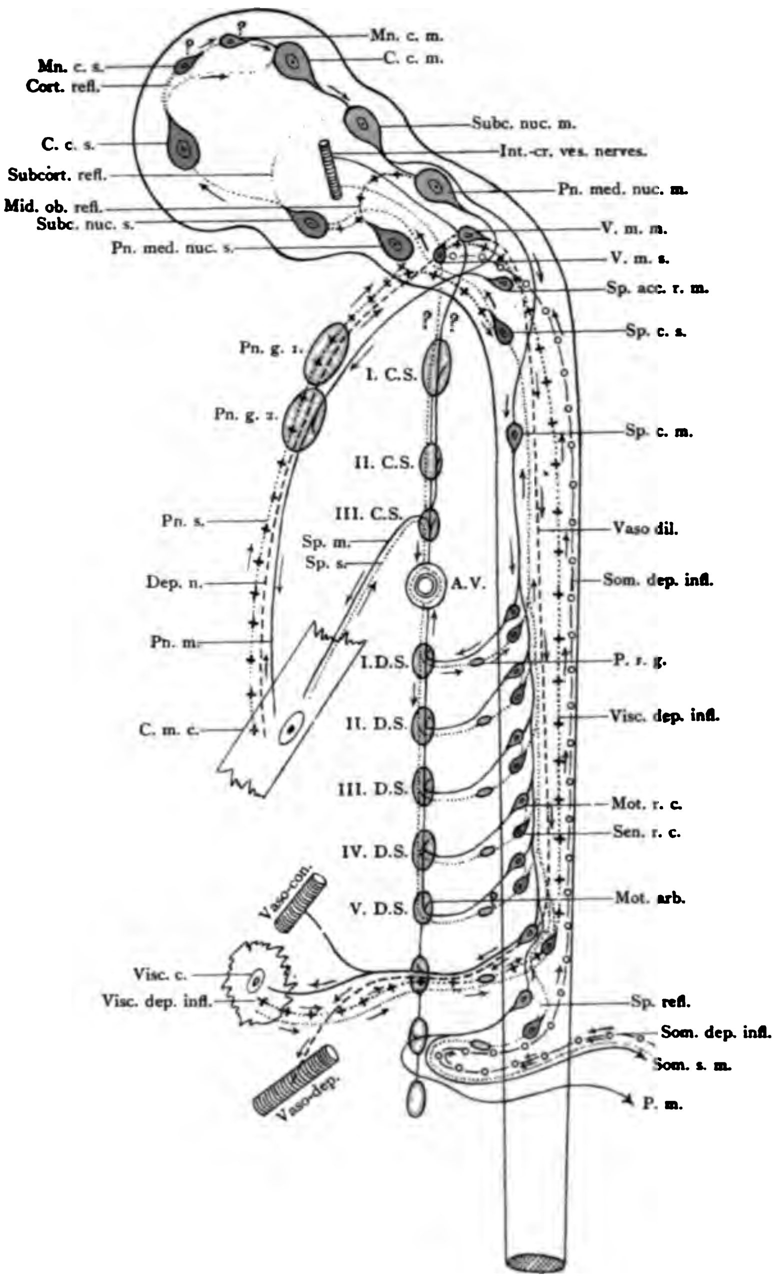
Mn. c. m.
C. c. m.
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Cort. refl.
Subc. nuc. m.
C. c. s.
Int.-cr. ves. nerves.
Subcort. refl.
Pn. med. nuc. m.
Mid. ob. refl.
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Vaso dil.
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Sp. s.
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Dep. n.
A.V.
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I.D.S.
Visc. dep. infl.
C. m. c.
II. D.S.
III. D.S.
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Sen. r. c.
IV. D.S.
Mot. arb.
V. D.S.
Vaso-con.
Visc. c.
Sp. refl.
Visc. dep. infl.
Som. dep. infl.
Som. s. m.
Vaso-dep.
P. m.

FIG. I.—DIAGRAMS OF VISCERO-NEURAL RELATIONS OF THE HEART.

Mark of interrogation attached to any letter indicates the uncertainty of the fact; pointed-out arrows indicate the direction of the travelling influence. C.m.c., Cardiac Muscle Cell. Pn. m., Motor portion of Pneumo-gastric Nerve. Pn. s., Sensory portion of Pneumo-gastric Nerve. Pn. g. 1, Root Ganglion of the Pneumo-gastric Nerve. Pn. g. 2, Trunk Ganglion of the Pneumo-gastric Nerve. Dep. n., Depressor Nerve of the Heart. Pn. med. nuc. s., Sensory Medullary Nucleus of the Pneumo-gastric. Pn. med. nuc. m., Motor Medullary Nucleus of the Pneumo-gastric. Mid. ob. refl., Reflex in middle Oblongata. Subc. nuc. s., Subcortical Sensory Nucleus. Subcort. refl., Subcortical Reflex. C. c. s., Sensory Cortical Cell. Cort. refl., Cortical Reflex or Reaction. Mn. c. s., Sensory Mnemonic Cell. Mn. c. m., Motor Mnemonic Cell. C. c. m., Motor Cortical Cell. Subc. nuc. m., Motor Subcortical Nucleus. Int.-cr. ves. nerves, Intra-cranial Vescular Nerves. V. m. m., Motor Cell of Vaso-motor Centre. V. m. s., Sensory Cell of Vaso-motor Centre. Sp. acc. r. m., Motor Root of Spinal Accessory Nerve. Sp. c. s., Sensory Cell in Spinal Cord. Sp. c. m., Motor Cell in Spinal Cord. Vaso-dil., Vaso-dilator Tract. Som. dep. inf., Somatic Depressor Influence. P. r. g., Posterior Root Ganglion. Visc. dep. infl., Viscero Depressor Influence. Mot. r. c., Motor Root Cell. Sen. r. c., Sensory Root Cell. Mot. arb., Motor Arborescence in Sympathetic Ganglion. Sp. refl., Spinal Reflex. Som. s. m., Sensory-motor Somatic Nerves. P. m., Pilo-motor Nerves. Vaso dep., Vaso Depression. Vaso. con., Vaso Contraction. Visc. c., Viscerial Organic Cell. Sp. m., Spinal Motor Nerve of Heart. Sp. s., Spinal Sensory Nerve of Heart. I. C.S., First Superior Cervical Ganglion. II. C.S., Second Cervical Ganglion. III. C.S., Third Cervical Ganglion. A.V. Rings of Vicusseus. I. D.S.-V. D.S., First to Fifth Dorsal Sympathetic Ganglion.

Cerebro-Spinal Sensory Tracts marked in dotted lines. . . .+·····+·····+·····+
Cerebro-Spinal Motor Tracts marked in uninterrupted lines.-------------
Other Tracts specially marked thus . . . .•—•—•—•

division into distinguishable chambers, is in reality a complex single tube with communicating orifices, and the systemic circulation is to all intents and purposes a rhythmically pulsating vessel propelling blood supplied to it, as a rule, from a higher level than that which it occupies itself, and always under a steady materno-placental pressure which influences it. The active and passive phases of its action are therefore practically equal. The "tic-tac" of fœtal cardiac action shows little distinction between the duration of systole and diastole. Excessive uterine pressure from interference with the oxygenation of the fœtal blood may indeed diminish the normal rapidity of the fœtal heart, but its two chief phases are of nearly if not quite the same duration. With the introduction of the second or pulmonary circulation at birth, the closure of fœtal communications, and the withdrawal of placental pressure, the increased activity of the organ requires a greater period of rest, and a noticeable distinction between the duration of systole and diastole arises, which increases with the growth of the organism, until, in adult life, the duration of diastole is distinctly greater than that of systole in the normal and unaccelerated cardiac cycle.

Under what are usually regarded as abnormal circumstances, the duration of both phases of the cycle may be equally increased or equally diminished, or one or other phase may be increased or diminished in proportion to the complementary action. Thus arise the conditions of abnormal slowness of the heart's action or bradycardia, abnormal rapidity of the heart's action or tachycardia, and irregularity in the phases of the cycle or arrhythmia. Moreover, in this arrhythmical action there may be a certain periodicity of variation which introduces a rhythmical form of arrhythmia, to use an apparently contradictory phrase, by means of which certain recognised variations are brought about, such as the bigeminal and trigeminal pulse, and the associated coupling of beats in the heart's action. Such abnormalities may be more or less persistent or even permanent, and then they become so marked a clinical feature that the apparent causes and consequences of the condition are capable of separate study as pathological entities, and are so considered in many text-books, although it is questionable whether any of them have a right to be regarded as such. Taking them *seriatim*, we shall first consider abnormal retardation of the heart's action.

Bradycardia.—Grob, who was the first to apply the term bradycardia to the condition originally described by Adams and Stokes, found in 3578 cases examined as to pulse-rate that an unusually slow pulse existed in six cases. In about 0·17 per cent of these cases, therefore, the condition was to be regarded as physiological. The pulse-rate in males is on the whole slower than in females, and all Grob's physiological cases occurred in men. Of persistent cases of bradycardia associated with signs and conditions which remove them from the category of the physiological, the majority also occur in men, although the condition is met with among women. In thirty-eight cases collected or observed by the writer, thirty-one occurred in males. The preponderance of this sign among men brings it statistically in line with the greater prevalence of angina pectoris in men as compared with women. As regards the influence of age, among the cases examined by the writer, one occurred in a boy 15 years of age, two between 20 and 30, two between 30 and 40, seven between 40 and 50, six between 50 and 60, eleven between 60 and 70, seven between 70 and 80, and one between 80 and 90. There was thus an increased frequency in the occurrence of the condition as age advanced, for the seventh decade may be regarded as the usual term of human life. Although the data upon which this conclusion is based are not numerous, it seems probable that bradycardia may be regarded as a "disease" of middle and later life and preferentially affecting the male. The alleged causes of bradycardia are cardiac overstrain from sudden or great physical exertion, alcoholism, syphilis, fibrosis of the heart, or fatty metamorphosis of the myocardium with or without associated arterio-sclerosis, and antecedent Graves' disease with tachycardia. Well-marked bradycardia of a less permanent kind has also been known to follow febrile movement and exhaustion, such as that which accompanies or is due to pneumonia and influenza. The changes which occur in the female organism after accouchement are also at times associated with slow pulse. A quasi-physiological form is known to occur after prolonged fasting or starvation, while reflex influences and some poisons, such as opium, and under certain conditions, digitalis, account for other cases. Finally, in a considerable number of reported instances, no cause could be assigned for even fatal cases of bradycardia, and the heart is stated, somewhat hastily, to have been perfectly healthy.

Symptomatology.—Like cardiac failure in general, persistent bradycardia may be of fairly long, or of short duration; it may be an acute or chronic process. Persistent bradycardia of short duration is of necessity fatal, otherwise it would come into the category of the transient form which sooner or later gives place to a normal pulse, and is due to temporarily debilitating causes. The fatal form of persistent bradycardia of short duration is in reality a recurrent syncope, and is not very frequently fully observed. When it is, however, it presents a very striking clinical picture. "At short intervals there is a sudden cessation of all pulsation—a complete and prolonged intermission in cardiac action—followed at first by slowly recurring pulsations, 18, 20, 30, 40 times in the minute, again followed by another complete cessation of pulse. This syncopal bradycardia may be varied by short periods of more accelerated pulsation up to 60 or 70 in the minute, followed again by a complete pause. The patient during these pauses may not lose consciousness and may be quite aware of their advent, which he may signalise by an alarmed shouting and attempts at deep inspiration. Respiration is generally accelerated, the adjuvant mechanism of breathing coming to the assistance of the heart. If unconsciousness supervene, it may be associated with an epileptiform seizure. These grave symptoms may eventuate in recovery, but frequently the attack recurs and ultimately syncope becomes complete and prolonged, and death closes the scene" (Morison, *Cardiac Failure*, p. 56, London 1897). A well-marked, acute and transient bradycardia may, however, as has been remarked, be observed after exhausting febrile conditions, but soon gives place to a pulse of normal celerity, and of low tension, which in time improves in tone with the general recuperation of the patient (Figs. 2 and 3).

Chronic persistent bradycardia may last for years, and is frequently associated with a systolic bruit at the aortic orifice due to atheromatous or sclerotic changes in that situation. The pulse may be slow and full, and in correspondence at wrist and heart (Fig. 4); or, a slow bigeminal pulse may have its dropped-beat phase so little marked as to be scarcely perceptible at heart or wrist, so as to convey the impression, without careful observation, that the pulsations are half as numerous as they really are (Fig. 5). True persistent bradycardia is inaccelerable by posture, exercise, or stimulation. The rate of respiration may be slightly diminished when bradycardia is equable and persistent, but is usually accelerated more or less when the condition is due to cardiac failure of a most acute character.

Diagnosis.—Bradycardia being a mere symptom is not to be distinguished from any other condition. But two points have to be borne in mind in examining such cases, namely, (1) the possibility of cardiac action and pulse-rate manifesting the sesquipedalic or dropped-beat phenomenon, a point which must be determined by an examination both of the heart and the radial pulse; and (2) the fact that bradycardia may be due to causes either within or outside the heart. To determine the latter point Dehio has ingeniously suggested that atropin may be employed as a test. In intrinsic, or, as Dehio terms it, cardial bradycardia, atropin fails to accelerate the heart, while in extrinsic or extra-cardial bradycardia it succeeds in doing so.

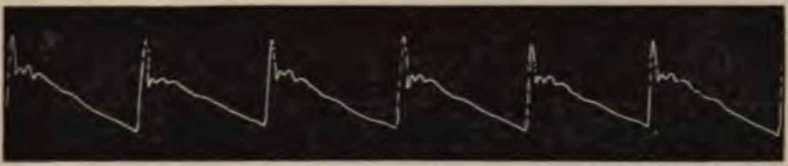

Fig. 2.—Bradycardia following pneumonia.

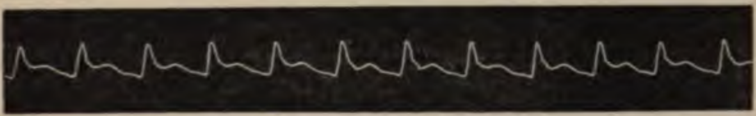

Fig. 3.—The same pulse soon after the bradycardia had disappeared.

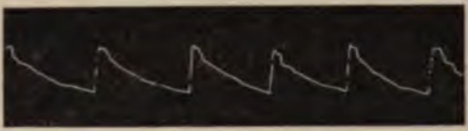

Fig. 4.—Chronic persistent bradycardia.

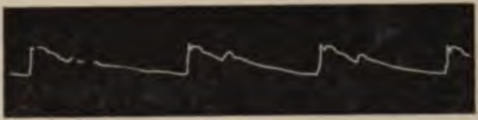

Fig. 5.—Bradycardia with dropped beat.

Pathology.—The pathological conditions which have been found in association with bradycardia are proliferation of the interstitial tissue of the heart, fatty degeneration of its muscle cells, atheromatous changes at the aortic orifice with, in many cases, systolic bruit, and likewise, in many cases, in association with a general arterial thickening, usually due to senile change. The defective quality of blood so frequently found in connection with fatty degeneration of the heart and various cardiac neuroses may also be present when the neurosis takes the form of bradycardia. Visceral nerve changes, central and peripheral, have been little investigated. In Holberton's well-known case, the autopsy in which was made by Robert Liston, there was said to be some induration of the medulla oblongata, and enlargement of the pneumogastric nerves as well as of the middle cervical ganglion on the right side. In congested hypertrophic conditions of the heart Ott has found proliferative changes in the cardiac ganglia, and in dyscrasiæ due to anæmia and pyæmia, fatty change in the ganglion cells themselves, together with a like metamorphosis of the muscle cells. It is difficult, however, to attribute bradycardia to any one or to any combination of these changes, for some of them are even more frequently associated with tachycardial and arrhythmical conditions of cardiac action. Stokes of Dublin, who noted the frequent occurrence of bradycardia with obstructive atheromatous states at the aortic orifice, considered that the whole bodily conditions of which atheroma and arterial degeneration are the outcome, might be regarded as causal of slow pulsation. That the aortic lesion may have some influence in producing this state may, however, be argued from the comparative slowness and regularity of the heart's action in aortic as compared with mitral disease. Our ignorance of visceral neural pathology prevents the expression of any useful opinion as to the share taken in this matter by the nervous system, but there are circumstances which point to the abolition or depression of the accelerant action of the spinal cardiac nerves, however induced and associated with whatever anatomical changes, as the probable cause of retarded cardiac action.

Prognosis.—This in bradycardia due to fever, poisons, or sudden failure of the heart may, of course, not be gloomy. The effect passes off with the disappearance of the cause. But in persistent cardial bradycardia the prognosis is always grave. The usual termination is in death by syncope with or without associated epileptiform phenomena.

Treatment.—The treatment of bradycardia must be guided by the view taken of its most probable cause, the opinion formed as to its transient or persistent character, and the possibility of its being influenced by therapeutic agencies. Extra-cardial bradycardia must be combated by cardiac accelerants, of which belladonna, ether, and the nitrites are the chief. The bradycardia due to sudden dilatation or cardiac overstrain may be beneficially influenced by rest, the digitalis group of remedies, and in some cases by bleeding. Persistent bradycardia in elderly people, however, which, as a rule, is uninfluenced by posture or stimulants, calls for little treatment beyond that proper to senile arterio-sclerosis in general. Chief among these may be mentioned the employment of short courses of mercury, with or without the addition of digitalis or belladonna, and an occasional saline aperient. In many cases, however, a "masterly inactivity" is indicated. If the bradycardia be unassociated with subjective discomfort, it is wisest in many cases to avoid the use of specific cardiac agents, which may upset the balance, if they act at all, of what is in reality a form of established cardio-vascular compensation. The issue of such cases being as a rule syncope, emotional and physical stress, and the sudden increase of the vascular contents by the imbibition of large quantities of nutritive fluids, should be avoided.

TACHYCARDIA.—The term tachycardia was first applied by Gerhardt to a markedly quickened action of the heart which had been previously described in this country. Accelerated cardiac action may be met with in various degrees and continue for very varying periods of time. So-called "palpitation of the heart" is usually a transient acceleration of the heart's action, but being usually of short duration, and the acceleration in many cases not excessive, it has not been classed with tachycardia proper, of which at least three varieties exist. These are: (1) A considerable acceleration associated with certain nerve-lesions, to which reference will be made again; (2) A long-continued acceleration without such lesions; and (3) An excessive acceleration of comparatively short duration, arising suddenly and, as a rule, ceasing as suddenly, and usually associated with a detectable degree of cardiac dilatation, to which Bouveret in 1889 applied the term essential paroxysmal tachycardia.

The influence of sex is more evenly divided between the male and female in tachycardia than in bradycardia, and its commencement or occurrence in earlier life than the latter condition has also been remarked. The condition may, however, be met with in its most typical and paroxysmal form in comparatively advanced life, as in the fifth or sixth decade. These facts appear to argue its essentially neurotic character, as the nervous system of the child and of woman are more mobile than that of man as a rule, and the catabolic or quickened action of the heart is, on the whole, a more frequently-witnessed physiological phenomenon than anabolism or retardation. Neurasthenia, or that gradual and general loss of reserve force in the nervous system which is usually associated with exaggerated reflexes, mental and bodily, is particularly conducive to paroxysmal outbursts of accelerated cardiac action. Cardiac overstrain may, in this case, as in the case of other forms of disordered cardiac motion, be an important causal factor. External agencies, such as belladonna and tobacco, may likewise induce the condition. Tachycardia is indeed of the belladonna type of poisoning, as bradycardia is of the opium type. Certain cardiac valvular defects likewise conduce to the occurrence of tachycardia. It has been remarked that bradycardia may be associated with aortic lesions, and that aortic lesions are generally associated with less acceleration than mitral lesions. The mitral type of cardiac action is, on the other hand, tachycardial, and we shall learn that cardiac dilatation, with mitral insufficiency, is not infrequently observed in paroxysmal tachycardia. The fact that one of the functions of the pneumogastric nerve is cardiac inhibition, and the fact, likewise, that pressure upon these nerves by neoplasms or other growths is frequently associated with a measure of tachycardia, has led to the maintenance of the "vagus theory" of the condition, the action of that nerve being assumed to be more or less hampered, or in abeyance, under these circumstances. It must be remembered, however, that both physiologists and surgeons have shown that injury of one of these nerves has little permanent effect upon the heart's action, while injury of both is usually soon fatal. Herringham inclines to the opinion that not the nerve as a whole, but its peripheral endings, are at fault in tachycardia. Others have regarded it as an affection of the sympathetic nerves of the heart, and yet others as due to disease of the myocardium. Regarding the muscle cell as the ground common to the nerve-endings both of the vagus and sympathetic nerves, and as having a rhythmicality in a measure independent of either, it seems possible that there may be an emancipation of cellular rhythmicality from all nerve control in excessive paroxysmal cases; while in others, characterised by acceleration and augmentation, or by increased force with less rapidity, that the catabolic nerves may be active agents in producing the condition, the action of the retardant nerves being for the time in abeyance, but accumulating that energy which at last suddenly asserts itself in the restoration of slower action to the heart. We must be on our guard, however, not to throw the reins on the neck of imagination when we enter that area of neural exposition which has been termed the refuge of the destitute in search of theories.

The occurrence of such disorders in children whose organic cells are presumably normal, and the probable dependence of organic irregularities in some cases on reflex causes outside the organ most notably affected, seem to leave the initiative in such processes to the nervous system. Hence the justification for regarding these conditions as neuroses.

Symptomatology.—An accelerated palpitation of the heart with a sense of "fluttering" in the præcordial region, coming on without observable cause or after slight exertion, is a common phenomenon in anæmic persons, usually young, and as usually females. While this common condition is not that which has been raised to the dignity of a disease by the style and title of tachycardia, it is, nevertheless, a humble attempt in that direction. Could it succeed in persisting longer—for days, months, or it may be years, with an average pulse-rate of 120 or 130 in the minute, without other observable disorder, the condition would constitute persistent tachycardia. This state may for long be associated with little subjective sense of physical discomfort or general functional inactivity, and be indicated by no other fact than a persistent rapidity of pulse, perhaps accidentally detected. In course of time, however, even these cases tend to give out, and the tachycardiac patient becomes unfitted for the exertion hitherto undergone with apparent impunity. Functional disturbances of secretion and excretion manifest themselves, and sleep, the great recuperator of the nervous system, may become defective and unrefreshing. If, as may happen, organic disease of the nervous system, peripheral or central, and due to intrinsic changes or external pressure, be associated with the tachycardia, the evidences of these may in time become more apparent. The third and highest degree of tachycardia, being associated, after much shorter duration, with considerable subjective discomfort, is that which Bouveret termed essential paroxysmal tachycardia. The pulse-rate is usually about 200 in the minute, and can only be counted by auscultation of the heart (Fig. 6). It may continue for periods varying from a few hours to several days, and the longer the duration of the attack, the greater the probability of the occurrence of retrograde phenomena, such as congestion of the lungs, enlargement of the liver, and albuminaria. It is indeed probable that attacks of tachycardia, unassociated with much or any subjective sense of palpitation, may cease and only leave the retrograde phenomena as evidence of their recent occurrence. It is in this way that the writer is inclined to explain the occurrence in a patient of his of consolidation of the base of the right lung, prune juice expectoration, and very slightly-increased respiratory rate, but without either pyrexia or acceleration of pulse. The patient, a lady about thirty years of age, informed him that she had felt faint and giddy the day previous to his detecting these signs, but had had no other symptoms pointing to her condition. She was of a neurotic constitution, and suffered much from urticaria.

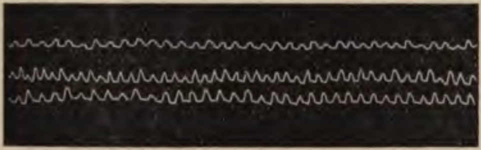

FIG. 6.—Radial pulse of essential paroxysmal tachycardia.

Herringham observed in a girl of eleven years, the subject of an attack of essential paroxysmal tachycardia, that the apex-beat was more visible than usual, in addition to the rate of pulsation being greatly increased.

Percussion usually shows the cardiac area in these cases to be increased in extent, and the sphygmograph declares the small and quick pulse to be one of low tension. Prior to the occurring of secondary stasis, there may be no subjective distress beyond a general sense of weakness, and the attack may subside as suddenly as it originates, leaving a pulse of normal tension and rapidity (Fig. 7). Herringham remarks that the attack often terminates during sleep, but that it has been determined to continue unabated during sleep in persistent cases.

Diagnosis.—The transient tachycardia so frequently met with among anæmic patients is easily distinguished by the presence of other signs of anæmia, and by the removal under treatment of the symptom with the improved condition of the blood.

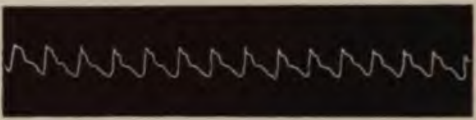

FIG. 7.—The same pulse as Fig. 6, after the attack had subsided.

A persistent tachycardia of moderate rapidity may be due to commencing Graves' disease, in which the classical signs of that condition have not as yet developed, or in which one or other of the striking phenomena in eye, or thyroid, or muscular tremor, are long delayed or altogether absent. Rapid arrhythmical pulsation of the heart may be distinguished from tachycardia proper by its very arrhythmicality, and the usually associated signs of arterio-sclerosis, which are frequently altogether absent in tachycardia, and also by the amenability of rapid arrhythmiæ to treatment. With essential paroxysmal tachycardia no other condition can be confused, nor is the degree of sudden cardiac dilatation associated with that state encountered in any other condition, in which the heart-muscle gives evidence of general soundness.

Pathology.—The transient tachycardia of anæmia has no recognisable physical basis beyond the characteristic alterations in the blood met with in that state, any more than has the neuralgia associated with the same condition. The hypersensibility conferred upon the sensory portion of the nervous system by anæmia, and manifested in some cases by neuralgia, is shown in yet others by disordered motor innervation, and by exalted irritability of rhythmical cells. In sustained tachycardia of moderate rapidity (100 to 150 beats in the minute) tumours in or pressing upon the pneumogastric have been met with in some cases, and in two mentioned by Martius both pneumogastrics were involved. As, however, in the latter cases the patients lived for some time in this condition, it is not probable that the conducting power of the nerves was altogether abolished. In bulbar paralysis, also associated with tachycardia, the pneumogastric nucleus has been known to be invaded by disease. Besides these changes, in five cases quoted by Herringham, there was fibrosis of the left ventricle in two, fatty degeneration of the myocardium in one, while in the remaining three dilatation was the only abnormality discovered.

Prognosis.—The prognosis in tachycardia, as in bradycardia, depends upon the nature of the cause. If this be removable the effects may also be expected to disappear. The cure of anæmia will also cure such tachycardia as is dependent upon it, the removal of sources of reflex tachycardia will likewise benefit the latter, but in a large proportion of the persistent cases the prognosis is not favourable, although the fatal issue may be delayed for a considerably longer period than is usually the case in "cardial bradycardia." The association of tachycardia due to any cause with organic valvular disease of the heart renders the prognosis both of the neurosis and the mechanical lesion doubly unfavourable, and the end may be sudden and syncopal, or more gradual and associated with all the retrograde venous phenomena of progressive cardiac failure.

Treatment.—In the treatment of the tachycardia symptomatic of anæmia, the use of arsenic is advisable, in addition to any other hæmatinic, such as iron, because there is reason in the belief that it has a tonic influence upon the nervous system. The peripheral palsies, induced by its over-use, argue a certain stimulating influence on peripheral visceral nerves when it is carefully employed. The drug treatment of persistent or paroxysmal tachycardia has, however, not been found to be effectual. Nevertheless, pharmaceutical agents calculated to support the three factors in organic action—the blood, the nerves, and the muscle cell—should be carefully and persistently employed, in conjunction with such hygienic measures as it is within the power of the patient to avail himself of. The representatives of this line of treatment may be said to be arsenic, iron, the digitalis group of remedies, the bromides, and, in the instance of cases of specific origin, the iodides and mercury.

In paroxysmal cases associated with valvular lesions, and which are not, of course, as a rule, cases of "essential tachycardia," much may be done, as we all know, by the recognised treatment of the lesion, for particulars of which the reader is referred to p. 427 *et seq.* The effect of opium in some of these cases, the physiological influence of which is bradycardial, is very striking.

Among external agencies a word may be said about the employment of cold. The careful employment of cold, in the form of ice, to the head or præcordia, for such periods as the patient can tolerate it, is frequently of service. A course of Nauheim baths is at times said to be of service, but the addition to this of gymnastic exercises, whether administered manually or by mechanism, is not to be undertaken without the closest supervision by the physician himself. Certain positions of the body, accidentally or instinctively assumed by the patient, appear at times to bring about improvement. Thus, compression of the thorax and abdomen and flexion of the legs on the trunk, have appeared to be of service, and probably act by raising the peripheral blood-pressure. Herringham recommends that pressure on the vagus should be always tried, "although it seldom succeeds." It is difficult, indeed, to see why it should succeed unless both vagi be compressed. We know that intermission of the heart's action may at times, and usually under special circumstances, be induced by compression of the vagus, but a vigorous and bilateral employment of this method does not seem to be free from the possibility of injurious interruption of the cardiac cycle. The treatment of known sources of reflex stimulation of the heart's action, such as local inflammatory disease of any kind, and displacement of organs, such as floating kidney, is indicated. The removal of adenoids is reported to have been curative in one case (Spencer Watson).

Having said so much, however, it ought to be added that it seems as impossible to retard the action of the heart in many cases of tachycardia, persistent and paroxysmal, as it is to accelerate it when bradycardia is of long duration. They appear to be what Dehio would call cardial, not extra-cardial, in origin, and as such, unaffected by the agencies which, under other circumstances, influence the action of the heart.

ARRHYTHMIA.—Under this term are included all abnormalities of cardiac action not solely characterised by diminution or increase of the rate of pulsation. In some respects the term is misleading, for abnormalities in pulsation are frequently observed which, while they differ from the isochronism of normal action, are, nevertheless, rhythmical in their arrhythmia. To rectify this contradiction, however, would lead to a multiplication of terms, which it is advisable, so far as is possible, to avoid. An ideal nomenclature no doubt would convey not merely the recognition of the fact of a particular kind of abnormality, but likewise the physiological nature of the action in question, and this the sphygmograph enables us in some measure to do in these cases.

Using arrhythmia in its widest sense, the condition is met with at all ages from childhood to old age and in both sexes. Its persistence, however, for a sufficient length of time to entitle it to be regarded as a definite morbid condition, is most frequently met with in adult life, and especially after the meridian of life is past. That is, when the wear and tear of life leaves its traces in texture. The condition is more common than either persistent bradycardia or persistent tachycardia, and perhaps owing to this very fact a statistical estimate of its incidence at certain ages and in both sexes is difficult to obtain. The writer believes, however, that it may be asserted that persistent arrhythmia, unassociated with organic valvular disease of rheumatic origin, is more common in men than in women, and most common among those in the arterio-sclerotic period during and after the fifth decade. The arrhythmia associated with rheumatic valvular disease may of course be met with much earlier and at any age, although, inasmuch as the same forms of arrhythmia may be met with both in the presence and in the absence of valvular lesions, the latter cannot be regarded as essential factors in their production.

The nature of the lesion appears, however, to have a certain influence upon the character of the heart's action in its endeavour to cope with the mechanical difficulty produced by it. Thus a certain type of arrhythmia may be more common in one form of valvular lesion than in another. Just as the heart's action is usually more regular when compensation is lost in aortic than in mitral valvular disease, so the arrhythmia associated with aortic disease tends to be more rhythmical and of a slower rate than the delirious hyposystolic pulsation of a tachycardial type so often met with in mitral disease (Figs. 8 and 9). What applies to the aortic and mitral valves applies also to the pulmonary and tricuspid, although the opportunity of observing the latter is rarely afforded. There may therefore be said to be an aortic and mitral type of arrhythmia, the former having a greater leaning towards bradycardia and the latter towards tachycardia, with the result that the arrhythmia of necessity partakes of the nature of both, periods of retarded pulsation occurring irregularly in the midst of an unequal tachycardia. Outside mechanical causes, the same influences which provoke bradycardia and tachycardia have an influence in the production of arrhythmia. Gout in its protean forms—rheumatism, influenza, alcoholism, syphilis, venery, tobacco, near or distant local causes of reflex action, and interstitial sclerotic and myocardial changes, may be associated with this as with other forms of disordered cardiac action. The discrimination of these is, however, of practical importance, as will be insisted upon in its proper place. Finally, when predisposing causes are present, "shock" may originate a persistent arrhythmia. Nor need this surprise us when we reflect that both physical and mental shocks may induce a fatal syncope and indeed persistent disorder in organs other than the heart. Thus a neurotic patient of the writer's, when towards the end of his fifth decade and in good health, developed glycosuria as the result of the suicide of an intimate friend whose cremation he had to superintend in accordance

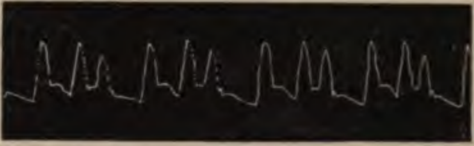

FIG. 8.—Rhythmical arrhythmia in aortic valvular disease with trigeminal pulse.

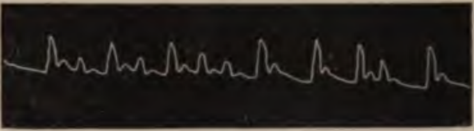

FIG. 9.—The arrhythmia of mitral disease.

with the wishes of the deceased, and about five years later, having the misfortune to be knocked down in the street by a bicycle going at good speed, the same subject developed a persistent and ultimately well-marked arrhythmia, which dated from that accident. In notes made prior to that event, his pulse was only on one occasion observed to be slightly intermittent (Figs. 10 and 11).

Symptomatology.—The inequality in *time* in the pulse of arrhythmia is usually associated with a variety in the *character* of the separate cycles in cardiac action and their corresponding pulse waves. In the simplest form of arrhythmia, indeed, the variation in time is more marked than in character (Fig. 10), but sooner or later alterations occur in this respect also. The pulse wave may then be characterised by a partial systolic rise which corresponds to a hyposystole at the heart, by a bold intercurrent systolic rise which denotes a hypersystole at the heart, or by a suppression of the systolic rise or intermission of the pulse, which denotes the condition of asystole at the heart (Fig. 11). All these may, moreover, be met with in the same pulse, and occur without determinable periodicity. The arrhythmia is then complete. Systole in varying force may, however, be associated, as has been stated, with hyposystole of regular recurrence, and give rise to the cardiac and radial pulse evidences of coupled and tripled beats (Figs. 5 and 8). In some cases the patient, when recumbent, evinces the coupled beat at the wrist and a quadruplication of sounds at the heart, which may be rendered phonetically as lūp—dŭp—lŭp—dūp, and on assuming the erect position, the heart's action being quickened, the pulse becomes regular at the wrist and the cardiac sounds may be rendered as lūp—lŭp—dūp. The acceleration of the heart in this case obliterates the first diastolic pause in the coupled beat, and the change is marked by an anachrotism of the pulse not previously present (Figs. 12, 13 and 14).

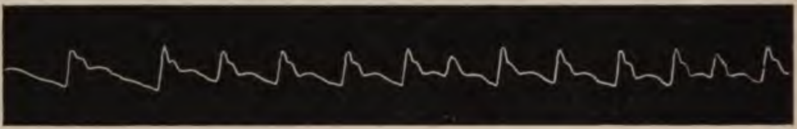

FIG. 10.—Incipient arrhythmia after shock.

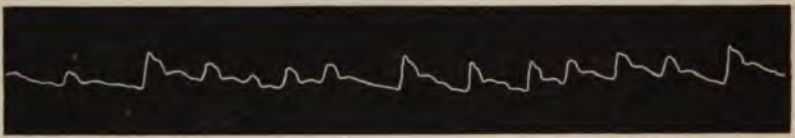

FIG. 11.—The same pulse four years later.

The objective signs of arrhythmia may be associated with no subjective discomfort on the part of the patient. On the other hand, a forcible irregularity of the heart may give the sense of palpitation, and well-marked intermissions are also frequently felt by the patient at the moment of their occurrence. The heart is felt to "stop," as patients at times remark. It is unnecessary to state that these objective and subjective signs may also be associated with other evidences of cardiac failure, local and general, such as valvular bruits, and various somatic and visceral forms of venous stasis. They may also manifest themselves without any of the notable signs of cardiac debility.

Diagnosis.—Arrhythmia, as its name denotes, is essentially *irregular pulsation.* It may be too quick a pulsation of the heart, with intervals of slower pulsation, and this *irregularity* is one of the means of distinguishing it from tachycardia. It is never likely to be confused with bradycardia, although in some cases, as has been stated, it may give place to a persistent domination of the latter sign. The diagnosis of tachycardial arrhythmia from persistent tachycardia may also be made by observing the effects of treatment by posture and drugs. The heart-rate of the former is more certainly reduced than that of the latter. The discrimination of tachycardial arrhythmia with functional bruit, usually soft and mitral systolic, from the same condition in association with organic valvular disease due to rheumatic endocarditis, is also important. As Huchard has pointed out in the former case, the bruit is a consequence of the disorder of cardiac motion and late in appearing, while in the latter the bruit is of early occurrence, and usually noted before the arrhythmical phenomena manifest themselves. In many cases, moreover, the functional bruit is due to mitral insufficiency from cardiac dilatation of a temporary kind, while the organic naturally persists, however much the heart may receive muscular compensation. Indeed, the more compensated the heart becomes, the more audible, in many organic cases, is the bruit.

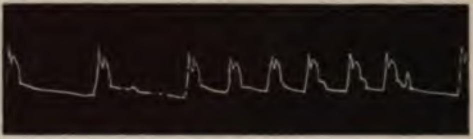

FIG. 12.—Arrhythmia with asystole.

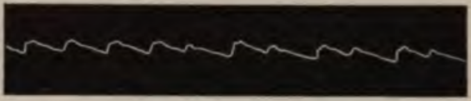

FIG. 13.—Arrhythmia with quadruplication of heart sounds when recumbent.

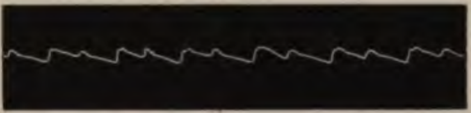

FIG. 14.—Another of the same.

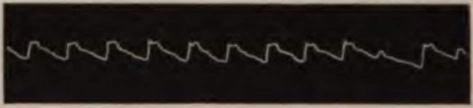

FIG. 15.—Triplication of heart-sounds in the same patient when erect.

Pathology.—The ultimate disappearance of arrhythmia in many cases, and at various periods of life, leads to the conclusion that it may sometimes depend upon transient conditions affecting one or more of the three factors in organic action, which do not leave permanent and recognisable traces in the heart. As, however, a large proportion of persistent cases occur within the arterio-sclerotic period of life after the fifth decade, it is not surprising to find that interstitial and myocardial changes, fibrosis, pigmentation, fatty degeneration of the heart, together with more general arterio-sclerosis, are associated with many cases of persistent arrhythmia. Inasmuch, however, as all these states may occur in an aggravated degree, and with every phase of cardiac failure, without the patient manifesting arrhythmical action of the heart, it follows that causes still largely unexplained lie at the bottom of the disordered action of the heart.

Of the intimate pathological changes in cardiac innervation, and in the innervation of other viscera, little more is at present known than has been already mentioned, but the more systematic examination of this point after death, and the more regular examination of the blood in such cases during life, are calculated to afford useful and much-needed information. There can be little doubt that the toxins generated by the germs of such infectious diseases as influenza may exact a morbific influence upon the visceral innervation, although of a less easily detectable character than the gross neural changes met with after diphtheria.

Prognosis.—Reflex and toxic arrhythmia may disappear at any age with the disappearance or removal of the cause, as likewise may the arrhythmia of cardiac overstrain and general exhaustion when these states arise in comparatively young people with a fair reserve of recuperative energy. The arrhythmia of the arterio-sclerotic age is, on the other hand, usually persistent and progressive, inducing cardiac dilatation and other evidences of cardiac failure, and tending to shorten the life of the patient. Under appropriate treatment, however, much may be done to prolong life in these cases, even when the underlying pathological condition is in an advanced stage, and the signs of cardiac failure well marked.

Treatment.—In this as in other forms of disordered cardiac action the removal or restraint of sources of reflex irritation is indicated. In the case of the young these will usually be found to lie in the gastro-intestinal tract and will be amenable to the usual dietetic and medicinal agents effectual in securing due digestion in and evacuation of the canal. In other cases, the treatment of a general condition of which the arrhythmia is but a symptom, is indicated. Thus, in the writer's experience, a case yielded to the use of thyroid extract in the case of a lady, the subject of indistinctly-marked myxœdema. The gout, glycosuria, or rheumatic heart affection associated with arrhythmia calls for treatment on recognised lines, for particulars of which the reader is referred to other portions of this work.

If arrhythmia be associated with obesity or neurasthenic conditions suggestive of impaired muscular power, a judicious alteration of rest and exercise, with or without salt baths, on the Nauheim principle, may be of much service. It is, however, in cases of arrhythmia associated with arterio-sclerosis that careful management may effect much. In these the Nauheim system, although often employed, is of little service, and not devoid of danger. These cases, in a large proportion of mankind, need cardio-vascular rest, not exercise, but rest need not be synonymous with absolute quiescence, unless well-marked evidence of cardiac failure be present. A regulation of the blood weight by prescribing small and easily digestible meals, and the avoidance of more than a minimum of alcohol and tobacco, together with gentle exercise, may do much among the better classes to promote efficient cardiac action. The question as to the advisability of absolutely prohibiting the use of tobacco, frequently arises. In the case of those who have undeniably over-smoked themselves, it may be as necessary to do so as it may be to absolutely prohibit the use of alcohol to the drunkard. But the rule of thumb and oracular prohibition of these articles may, in the case of those accustomed to their moderate use, do more harm than good. Although the will cannot influence the heart, the emotions can, and a large number of people cannot reconcile themselves to the abandonment of these articles without an amount of subjective worry, which it is desirable to avoid when the controlling influence of mental calm is very desirable. The poor overworked arterio-sclerotic patient, with arrhythmia on the other hand, frequently does well while in hospital on ordinary diet, rest in bed, and two to three ounces of brandy daily. He needs feeding, rest and warmth to strengthen his heart and to overcome his peripheral vascular resistance. Among drugs in such cases, if one be more valuable than another, it is mercury, given alone, or, if there be evidences of cardiac dilatation and failure, in combination with digitalis. Old masters, such as Stokes of Dublin, knew well the value of this combination, and that value will be found not to have been exaggerated by those who will follow their directions in the present day.

The proper use of mercury in arterio-sclerosis seems, indeed, for a time to have been largely a lost art. With the concurrent use of bland unirritating form of food, and the avoidance of acid condiments such as vinegar, however, mercurials may be given for a considerable period with benefit. In arterio-sclerotic cases with arrhythmia and cardiac failure, the well-known combination of blue pill, digitalis, squill, and hyoscyamus, often known as the "Guy's Pill" (but which is really attributable to that worthy physician, Dr. Matthew Baillie, who had no connection with Guy's Hospital), acts in many instances with the efficacy of a specific. The combination, indeed, often succeeds when the ingredients given separately fail, as Dr. Baillie maintained, for, while the mercury relieves the peripheral resistance to the circulation, the digitalis urges the weakened heart to more efficient action.

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IN commencing the subject of congenital malformations of the heart we cannot do better than begin with a classification of its various forms taken from Professor Vierordt's admirable monograph. This list gives some idea of the complexity of the subject and of the extraordinary multiplicity of forms which congenital defect of the heart may assume. In estimating this we must further bear in mind that it is much commoner to find several malformations combined in one heart than to meet with one only, and that in the case of patients who have attained adult age there are apt to be further complications as the result of post-natal endocarditis having attacked the abnormal parts.

Vierordt's classification is as follows:—

1. Patent foramen ovale, uncomplicated defects of the inter-auricular septum.
2. Uncomplicated defects of the inter-ventricular septum.
3. (*a*) Primary inflammatory stenosis of the ostium and conus of the pulmonary artery without malformation (with patent foramen ovale).
   (*b*) Stenosis and atresia of the pulmonary artery.
   (1) Without septal defect and transposition of the vessels.
   (2) With septal defect and transposition of the vessels.
4. Transposition of the large arterial trunks (with various complications).
5. Dilatation of the pulmonary artery.

6. Persistence of the truncus arteriosus (including cases of direct communication between the aorta and pulmonary artery).

7. Congenital stenosis and atresia of the commencement of the aorta (conus and ostium).

(*a*) From arrest of development.

(*b*) From fœtal endocarditis.

8. Congenital abnormalities of the semilunar valves.

9. Stenosis of the aorta near the entrance of the ductus arteriosus, persistent isthmus aortæ.

10. Congenital narrowness of the aortic system.

11. Patency (uncomplicated) of the ductus arteriosus.

12. Primary defects at the right auriculo-ventricular opening—

(*a*) Developmental.

(*b*) From fœtal endocarditis.

13. Primary defects of the left auriculo-ventricular opening—

(*a*) Developmental.

(*b*) From fœtal endocarditis.

14. Misplacements of the heart.

15. Malformation of the pericardium.

Causation.—The lesions of congenital heart disease may be divided into three groups according to their causation.

(1) In some cases the abnormalities in the heart are evidently of such a nature that they must have resulted from intra-uterine endocarditis. When this disease occurs in fœtal life it may be of the warty variety, especially when it affects the semilunar valves of the arterial openings. In the great majority of cases, however, it assumes the sclerotic form, and leaves behind it an opaque white or buff yellow thickening of the endocardium. It may cause contraction of one or more of the cavities or of the valvular openings, or shrinking or adhesion of the valves themselves. The condition occurs more frequently on the right than on the left side of the heart.

(2) In many cases, part or the whole of the lesion consists in persistence of openings in the inter-ventricular or inter-auricular septa or of the ductus arteriosus. Abnormal patency of any of these structures is referable to obstruction to the course of the circulation occurring at a period earlier than that of their natural closure. This obstruction may exist in the lungs or in the course of the pulmonary artery, in the right ventricle or at the tricuspid aperture; and it may equally well be situated on the left side of the heart either at the mitral or aortic orifice. The presence of a patent inter-ventricular opening indicates an earlier date for the commencement of this obstruction than an open foramen ovale or ductus arteriosus only. Sometimes on examining the heart and lungs no lesion can be found which could have caused any obstruction to the circulation; in such cases it is probable that the block has been of a temporary nature and has disappeared.

(3) The remainder of the cases, which cannot be grouped in one or other of these categories, present other imperfections of development which show no trace of being due to inflammatory action. Such are transposition of the large arterial trunks, congenital abnormalities of the semilunar valves, absence of the pericardium, etc. The causation of these arrests or perversions of development is in many cases exceedingly obscure. The experiments of Geoffrey St. Hilaire and other more recent observers have shown that a variety of mechanical and chemical influences (such as violent shaking and the injection of chemical irritants or of pathogenic germs and their toxins)

acting on an egg often result in congenital malformations in the chicken. The causes which under ordinary circumstances produce a like result in the human embryo are as yet quite unknown. It is extremely probable, however, as Ballantyne has pointed out, that the same morbific influences which occasion endocarditis in the later stages of intra-uterine life give rise to developmental defects when they act during the earlier months. In other words, the morbid influence which in the young embryo produces a teratological result has a pathological effect when it acts on a fœtus which has so far developed as to have differentiated organs.

It is important, in connection with the causation of congenital heart disease, to draw attention to the fact that a great variety of external malformations due to imperfect development (such as hare-lip, imperforate anus, webbed fingers, etc.) are frequently found along with it, and that it has been pointed out recently that a curiously large proportion of "Mongolian" imbecile children present congenital heart lesions. This association with the other defects of development seems quite as marked in instances of fœtal endocarditis as in the so-called developmental cases.

Clinical Phenomena.—The symptoms met with in cases of congenital heart disease vary greatly according to the extent as well as the nature of the lesions present, and the degree to which these interfere with the circulation. In some few cases (*e.g.* in some of septal defect) there may be no symptom or physical sign that could lead to a suspicion of abnormality during life. In others (*e.g.* in many of patent ductus arteriosus) there may be nothing discoverable beyond a murmur. In the great majority of cases, however, we meet not only with murmurs and other abnormalities on physical examination of the heart, but also with more or less deep cyanosis, with chilliness of the extremities and concentration of the blood; and we also often find clubbing of the finger-ends. If the interference with the circulation is marked we find some considerable degree of debility, and there may also be a varying amount of dyspnœa, attacks of cardiac pain, epistaxis, and epileptiform seizures of various types. The coincident occurrence of external defects of development has been already alluded to.

Some of these clinical phenomena deserve further mention.

Physical Signs.—The physical signs of congenital heart disease discoverable on palpation, percussion, and auscultation consist of alterations in the position or force of the heart's apex-beat, thrills, increased size or altered contour of the cardiac dulness, murmurs, and changes in the loudness of the sounds—especially of the pulmonary second sound. The combination of these phenomena met with has often much more significance than their individual occurrence. Thus a loud systolic murmur, with no increased dulness and no accentuation of the pulmonary second sound, has a very different meaning from an otherwise identical murmur accompanied by the usual signs of cardiac hypertrophy.

The murmurs are usually peculiar in their areas of audition, and in their lines of propagation, which do not correspond to those characteristic of any valvular lesion. It is often difficult, if not impossible, to determine the area of their maximum intensity, and this may also vary from time to time in the same case. In the very great majority of instances the murmurs are systolic in time; rarely, however, they may be diastolic or presystolic. Peculiar humming sounds are sometimes met with, especially over the base of the heart. It is to be remembered that murmurs due to congenital cardiac defects may change considerably in character and distribution if the patient becomes anæmic.

Cyanosis.—Cyanosis is present in the majority of cases of congenital heart disease, and it is so characteristic of these cases that they have been spoken of as instances of "morbus cœruleus." The discoloration varies greatly in degree. When well marked it is deeper than that produced by almost any other pathological condition. It is visible all over the body, but is especially noticeable in the extremities (fingers, nose, ears, etc.), and on the visible mucous membranes. When severe it may be accompanied by some puffiness of the features, but œdema of the extremities is rare, and only occurs late in the course of the case. The cyanosis is usually present at birth and persists during life, but it may only come on when the patient is several years old. Its degree varies from day to day according to the general health of the patient.

The cyanosis of congenital heart disease differs from other forms of cyanosis in degree rather than in kind. It is important to remember, however, that cyanosis is not met with to any appreciable extent in childhood as a result of post-natal endocarditis; its presence always, therefore, indicates a congenital lesion (either developmental or from endocarditis).

The causation of the extreme cyanosis in these cases has long engaged the attention of the medical profession, and several explanations of it have been suggested.

(1) The view that it is due to admixture of the venous and arterial blood allowed by their defective separation in the abnormal heart is to be considered no longer tenable. Extreme cyanosis has been found in cases where the condition of the heart did not permit of any abnormal mixture of this kind; and, again, individuals have been known to live for years without a trace of cyanosis, although their hearts presented more or less extensive septal defects.

(2) A more widely accepted explanation is that first enunciated by Morgagni, that the discoloration is merely the result of congestion of the venous system arising from backward pressure. It seems probable that this accounts to some extent for the phenomenon, and the differences noticed between the cyanosis of ordinary heart and lung cases and that of congenital malformation may be partly at least attributable to the very early onset of the backward pressure having caused greater dilatation of the smaller blood-vessels. The fact that the blood which distends these is abnormally dark from its concentration must also be borne in mind.

(3) Dr. Lees has presented strong arguments in favour of the cyanosis in congenital heart cases being simply an index of the extent to which aeration of the blood in the lungs has been hindered, the discoloration being due, not to venous congestion pure and simple, but to the congestion of non-aerated blood.

Clubbing of the Fingers and Toes.—This is a common symptom of congenital heart disease; but the exact conditions under which it occurs are not quite clear. It is present in most cases where the cyanosis is extreme, but not in all, and it may be present without any cyanosis. Dr. Lees points out that it is not likely to occur if the systemic venous congestion is prevented by the presence of a widely patent foramen ovale. When clubbing of the fingers and toes is present to a marked degree a corresponding condition of the nose and ears is usually seen.

Blood.—In cases of this form of cyanosis, as well as in others, there is a concentrated condition of the blood. Its specific gravity is increased (1070-1080), and the number of coloured blood corpuscles may reach as high as from 8 to 9,000,000 in the cub. cm., while the hæmoglobin may be over 150 per cent.

Some Forms of Cardiac Malformation.—A few facts may now be given regarding some of the varieties of congenital heart lesion, including those which are most frequently met with in clinical work or in the post-mortem room.

Patent Foramen Ovale.—Very little blood passes through the foramen ovale at birth, although the opening is often not completely closed before the middle of the first year. Not uncommonly the foramen persists during life in the form of a small valvular opening. This scarcely amounts to an abnormality, and gives rise to no symptoms. When, however, the opening is large and not valvular, it is to be regarded as distinctly abnormal, even if, as sometimes happens, it has no apparent effect on the health. In some cases the patent foramen is accompanied by a more or less extensive defect of the adjacent inter-auricular septum. Patency of the foramen ovale is not very rarely the only malformation discoverable; it is, however, much more common to find it along with other malformations of the heart or large vessels.

The symptomatology of this defect is still very obscure. Frequently it gives rise to no symptoms or physical signs at all. Such symptoms as have been ascribed to it are inconstant and ill-defined. Murmurs of various kinds have been met with, and these have not only been presystolic or diastolic, as might have been expected, but quite as often systolic in time. The area of their maximum intensity has also varied, but they have generally been heard best about the level of the 3rd or 4th costal cartilages. In some cases there has been cyanosis; in others it has been absent. Under ordinary circumstances the diagnosis of these cases is impossible. In the rare instances, however, in which a patient with this defect acquires mitral incompetence in later life, the defective state of his inter-auricular septum may be betrayed by the occurrence of venous pulsation in the neck without evidence of tricuspid disease.

The prognosis in these cases is obscure. Many of the patients die in early infancy, but not a few instances are on record of individuals with large defects of the inter-auricular septum who have reached adult life and even old age (sixty to eighty years). It must be remembered, however, that the presence of an open foramen ovale constitutes a source of danger to life in cases in which anything of the nature of venous thrombosis is present. A detached fragment of a thrombus carried to the right side of the heart is very apt to pass directly into the left auricle, thence through the left ventricle into the arterial system, causing an embolism in the brain or elsewhere.

Defects in the Inter-ventricular Septum.—The normal inter-ventricular opening closes by the eighth week of intra-uterine life. A permanent opening in this situation constitutes one of the commonest malformations of the heart. It may occupy the position of the "undefended space," or membranous portion of the septum, or it may be at one or other side of this area. It is but rarely situated near the apex. Although occasionally found alone, this malformation is generally one of several. It is especially apt to be associated with pulmonary stenosis. When it is met with to a very marked degree along with a large defect of the inter-auricular septum, we have the condition which has been described as "corbiloculare" or "reptilian heart."

The physical signs produced by defects in the inter-ventricular septum are a matter of great difference of opinion. It is generally believed, however, to produce a loud, harsh, long-continued systolic murmur, which is audible chiefly over the upper third of the præcordia, in front, and also markedly in the inter-scapular region behind, and which is not accompanied by any thrill. Many differences in the physical signs have, however, been described by good observers. Cyanosis may be absent; and there is no question that very large defects in the inter-ventricular septum may persist without causing any symptom or physical sign whatever.

The prognosis is, on the whole, less favourable than in cases of open foramen ovale. The presence of an inter-ventricular communication causes greater interference with the circulation, and leads at an earlier period to cardiac hypertrophy. Cases have, however, been recorded in which patients with this lesion lived forty and even forty-five years. The danger of the occurrence of embolism is the same as in cases of patent foramen ovale.

Stenosis and Atresia of the Pulmonary Artery.—This is probably the commonest congenital malformation of the heart.[1] It may be due either to abnormal division of the common truncus arteriosus in the course of development, or to the result of fœtal inflammation. The exact situation of the narrowing varies. It may be caused by thickened and adherent valves, or may be situated above the valves. The artery itself may be thickened, or there may be a diminution in size of the conus. The lumen of the vessel may be quite obliterated. It is usually accompanied by patency of the inter-ventricular or inter-auricular openings, or both, and often by an open ductus arteriosus.

The symptoms are generally striking. There is usually marked cyanosis, with its accompaniments. Often the right side of the heart shows distinct evidence of enlargement. The auscultatory signs vary considerably. A loud systolic murmur in the second and third left spaces near the sternum, which is propagated into the vessels of the neck, is what is most characteristic. There may, however, be no murmur at all. In cases of simple stenosis the pulmonary second sound is faint or absent, but in some cases, where the valves are not implicated and the ductus is widely open, it may even be accentuated.

The prognosis in cases of pulmonary stenosis is not good as regards the prospect of long life. In exceptional instances patients have been known to live more than forty years. The great majority, however, die in childhood or as young adults. In cases of atresia the chances of life are considerably less than in those of stenosis. There would seem to be a special tendency for patients with this malformation to suffer from various forms of cerebral disease. It is said, also, that they are peculiarly liable to tuberculosis of the lungs.

Transposition or Malposition of the Aorta and Pulmonary Artery.—This is a group of not very common cases which are complicated both as to their anatomical details and their mode of production.

The physical signs are also very obscure and equivocal. There is generally marked cyanosis, and systolic murmurs are usually present. These, however, may possibly be referable to the complications which are present. According to Hochsinger, the presence of extreme cyanosis, with pure heart-sounds and accentuation of the second sound at the base of the heart, gives sufficient ground for a diagnosis of this condition in an uncomplicated form.

The prognosis in cases of transposition of the large vessels is very bad. In a few instances the patients have survived to adult age, but in the great majority they die within the first six months of extra-uterine life.

Stenosis and Atresia of the Aorta.—This is a much less common condition than narrowing of the pulmonary artery. Most forms of it are inconsistent with continued extra-uterine life, and the children survive for a few days at most. The only variety of the malformation which is compatible with longer life, and therefore of clinical interest, is the stenosis or atresia which occurs near the entrance of the ductus arteriosus. This is situated at the commencement of the descending aorta, either above or below the ductus. The obstruction at this point leads to great dilatation of the aortic behind it and of its branches, and an extensive collateral circulation develops.

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[1] According to Peacock, more than four-fifths of the children with congenital heart disease who reach twelve years old suffer from this lesion.

The symptoms in these cases are either slight or quite absent in childhood, so that the condition is not diagnosable. They become, however, steadily more marked in later life. This is largely due to the fact that the constricted portion of the vessel remains about the same size, while the lumen of the rest of it grows with age, so that the effect of the disproportion between the two becomes increasingly felt. Cyanosis is seldom present. The physical signs which are characteristic of this malformation in adult patients are as follows:—(1) Marked hypertrophy of the left side of the heart; (2) a loud systolic murmur, accompanied by a strong thrill over the manubrium sterni to right of it, and in the jugular fossa, which is also conducted into the vessels of the neck; (3) a visible collateral circulation in the form of superficial, dilated, pulsating arteries, recognisable over the chest and abdomen, in which systolic murmurs may sometimes be heard. The diagnosis may be confirmed by the occurrence of retardation of the femoral pulse and marked weakness of the arterial pulses all over the lower half of the body, contrasting with those of the distended vessels above.

In this form of aortic stenosis or atresia the prognosis is much more favourable than it is in cases of constriction of the pulmonary artery. In some cases its presence seems to have been consistent with a long and apparently healthy life. When death occurs it is sometimes due to rupture of the aorta, sometimes to cerebral or pulmonary lesions.

Congenital Abnormalities of the Semilunar Valves.—Abnormalities in the number and size of the semilunar valves are not very uncommon. One segment may be unusually small, or there may be two or four instead of three. Such malformations are generally of themselves of no consequence. It is found, however, that in later life the abnormal valves are specially liable to be affected by endocarditis.

Patent Ductus Arteriosus.—The ductus arteriosus rapidly closes after birth, and it should be entirely obliterated somewhere between the tenth and twentieth days of life. Failure of this normal process of involution and consequent persistent patency of the canal is a common complication of various congenital malformations of the heart and vessels, as already mentioned. Occasionally, also, cases are met with in which an open ductus is the only lesion to be found. The persistent duct may be greatly dilated, in which case it often acquires a funnel shape, the aortic being the wider end. The pulmonary artery may be greatly dilated, and the left ventricle is generally hypertrophied.

When the lesion is uncomplicated there is no cyanosis until late in the progress of the case, and the patient often lives many years in the enjoyment of good health and without any abnormal subjective sensations. Cardiac hypertrophy and other indications of some embarrassment of the circulation are, however, apt to intervene sooner or later. The murmurs vary, but that which is much most frequently heard is a loud systolic bruit in the second left intercostal space a short distance from the sternum, which is accompanied by a palpable thrill and an accentuated second sound. Occasionally there is a diastolic murmur.

The prognosis in uncomplicated cases of patent ductus arteriosus is more favourable than in most forms of congenital heart disease. About half of the published cases have survived puberty, and many have had long and active lives without any signs of disease.

Acardia.—Acardia or absence of the heart is met with in rare instances of still-born twin monstrosities.

Double Heart.—A double heart has been described in cases of extremely deformed fœtuses. Such a condition is, however, never met with clinically.

Misplacement of the Heart.—The heart may be displaced to the right side, or forwards, or even, in very rare cases, upwards into the neck or downwards into the abdominal cavity.

Dextrocardia.—Misplacement of the heart to the right may occur without any corresponding malposition of the other organs. Usually, however, it is only part of a general transposition of viscera (*situs viscerum inversus*). When it is present alone this condition causes no disturbance of the general health, but it is often associated with other malformations.

A lesser degree of misplacement to the right, so that the heart occupies a mesial position in the thorax, such as is found at a very early period of embryonic life, is occasionally met with, usually along with extensive developmental defects.

Ectopia Cordis.—Ectopia cordis or prolapse forwards of the heart is a condition which dates from a very early period of intra-uterine life. It is associated with congenital fissure or entire absence of the sternum, and sometimes with defect of some of the ribs. It varies greatly in degree, and when severe may be associated with absence of the pericardium.

Absence of the Pericardium.—Absence of the pericardium may occur along with abnormalities in the position and form of the heart or independently of other defects. It may be complete or partial.

General Diagnosis of Congenital Heart Affections.—It is not generally very difficult in childhood to determine in any given case whether a cardiac lesion is congenital or acquired. The most important points to be considered in making the diagnosis are:—The presence of cyanosis; the loudness of the murmur taken along with the age of the child; an atypical situation and propagation of the murmur, and the presence of hypertrophy of one or other side of the heart.

In children who are weakly and anæmic it is always well to be particularly cautious about the diagnosis of congenital heart lesions from the presence of murmurs alone. In such cases comparatively loud basic murmurs may be found, which disappear entirely on recovery from the general condition of debility.

The diagnosis of the exact condition is a question which must, in the large majority of cases, remain unanswered owing to its extreme difficulty. As the patient grows older the diagnosis of the lesion becomes increasingly difficult owing to the great frequency of secondary endocarditis of the congenitally abnormal structures. Hochsinger's axioms as to diagnosis, which are founded on the observation of children under five years old, but which mostly apply equally well to older patients, are (slightly abridged) as follows:—

1. Loud, harsh, musical murmurs, with a normal or but slightly increased area of dulness, are met with in little children only in congenital cases. When acquired inflammatory heart affections occur in them, with very loud murmurs, they invariably cause great increase in the cardiac dulness.

2. The occurrence of murmurs along with greatly increased cardiac dulness and feeble apex-beat in young children is in favour of congenital

disease. The increased dulness depends mainly on enlargement of the right heart while the left is but slightly altered. On the other hand, acquired heart disease in children is accompanied by increased force of the apex-beat, because its effect falls first on the left side, while the dilatation of the right heart sets in later, and does not affect the increased strength of the apex-beat.

3. The complete absence of murmurs at the apex, while they are distinctly present in the region of the auricles and over the pulmonary orifice, is always an important element in the differential diagnosis, and is more in favour of septal defects or pulmonary stenosis than of endocarditis.

4. Abnormal weakness of the pulmonary second sound along with a distinct systolic murmur can only be explained, in early childhood, by assuming the presence of congenital pulmonary stenosis, and consequently is worth remembering as a point in the differential diagnosis.

5. Absence of a palpable thrill, in spite of very loud murmurs audible all over the præcordial region, occurs almost exclusively in cases of congenital septal defects, and this condition is therefore against a diagnosis of acquired heart disease.

6. Loud systolic murmurs (especially those accompanied by a thrill) which have their point of maximum intensity situated over the upper third of the sternum, and are unaccompanied by any symptom of marked hypertrophy of the left ventricle, are very important for the diagnosis of persistence of the ductus arteriosus, and cannot be explained by the assumption of endocarditis of the aortic valves.

Prognosis.—The prognosis depends on the age of the patient, the state of his strength, and the presence or absence of hypertrophy of the heart and of cyanosis. The character of the murmur is of small importance in this connection compared with the general condition. In young infants the prognosis must always be very guarded until there has been time to observe to what extent the cardiac abnormality interferes with the vital processes. If the cyanosis is marked, the child weakly, and the heart enlarging in spite of care, the outlook is very bad. If, on the other hand, the patient's nutrition and vigour have been tolerably well sustained for several years, if cyanosis and clubbing are absent or slight, and the heart little, if at all, enlarged, the chance of his reaching manhood may be regarded as fairly good.

In estimating the effect of a congenital heart lesion on the general health, it is important not to attribute to its influence debility arising from other causes. It is not very uncommon to meet with children whose general health has been neglected owing to their having a loud heart murmur, which was supposed to indicate an incurable cause for all their ill-health, but who rapidly respond to ordinary treatment of their other ailments.

Treatment.—Generally the only possible treatment consists in keeping the patient quiet and warm and attending to his nourishment. In cases where there are periodic signs of failure of compensation (increasing dyspnœa, epistaxis, epileptiform attacks, etc.), great advantage is derived from confining the patient to bed for a time and giving digitalis or strophanthus. On the habitual cyanosis of these cases cardiac tonics have no effect. For cardiac pain and breathlessness, when severe, nothing but morphine, and that sometimes only in large doses, brings any relief. If rickets, anæmia, indigestion, or any other diseases are present, they must be carefully attended to.

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Heart, Surgery of.—As surgeons have advanced from the surgery of the pleura to the surgery of the lung, so the advance has now been made from puncture or incision of the pericardium to suture of a wound of the heart, and even to ligature of a bleeding coronary artery. The necessity for such an operation will very seldom come, yet a surgeon ought to have in his mind how he would meet it.

To clear the ground, those cases may be set aside where a needle or a knitting-needle has been driven by accident, or in an attempt at suicide, into the heart. In these cases the rule is clear and unmistakable, that the *foreign body* should be at once removed. One end of it may be felt under the skin; or its exact position may be defined by the X-rays. Even though it be gone altogether inside the pericardium, yet it should not be left there, unless it be so small that it might not be found after incision of the pericardium. In most of the published cases, it has not been necessary to do more than incise the superficial structures; nor has any serious bleeding followed the withdrawal of the foreign body.

Again, cases of *gunshot wound* need not here be considered; for though a case of surgical interference has been published by Riedel, and another by Podrez, yet such an operation must ever be one of the rarest possibilities of surgery; nor did their methods of procedure differ in any material way from those that have been used in cases of punctured or incised wounds made with a knife.

It is these *stabs* over the heart that have offered most opportunity for treatment by operation—the cases of Farina, Cappelen, Tassi, Rehn, Parrozani, and others. And, before operating, the surgeon has to face the fact that some undoubted cases of wound of the heart have recovered without operation. The older methods of treatment, absolute rest, without moving or speaking, real "immobilization," ice applied over the heart, low diet, and copious venesection — these methods did sometimes succeed. Nothing but hourly watching, and seeing evidence of steadily increasing pressure on the heart, and blood accumulating in the pleura, and the patient going in spite of treatment from bad to worse, justifies operation.

In a case of stab-wound of the heart, it is not likely that the surgeon need be afraid of wounding the pleura. Practically, he may be certain that he will find it already opened, and with blood poured into it. The internal mammary artery he may find divided, or may divide it, or may not come across it. The incision through the skin and the muscles has been made different ways, according to the position of the wound; either a long curved incision, or a flap. The cartilages resected have been, in most cases, either the fifth, or the fourth and fifth together. And, by whatever method, the surgeon must work freely, through a space large enough to let him see what needs to be done.

It has been recommended that the wound in the pleura should be at once closed, and the pleura pushed outward, out of the way. But, in practice, the opposite has been done; the wound in the pleura has been freely enlarged, the blood and clots have been washed out so thoroughly as the case permitted, and the pericardium has been dealt with through the pleura. The surgeon has found the wound of the pericardium, with blood running out of it, has enlarged it, and secured its edges with catch forceps.

There are, of course, cases where the pericardium has been opened, and no wound found on the heart (Tassi, Williams, Dalton). In Mr. Mansell Moullin's case (*Lancet*, 1897, i. 314) the patient had been kicked over the heart; the pericardium was opened, found full of blood, and drained, and he made an excellent recovery. In a few cases a wound of the heart has been seen, but so small as not to need suture.

In other cases the wound has been sutured with fine silk and a fine curved needle. From one to four sutures have been placed; and the ends of the first suture have been lightly held, to make it easier to pass the next. Cappelen passed, and tied, his sutures during the systole of the heart; Rehn, during its diastole.

In some cases the pericardium has been drained; in some, it has been closed at once. The hæmothorax may give rise, many days afterward, to the need of an operation to drain the pleura.

LITERATURE.—The whole subject of these cases, with full references, is discussed, with admirable clearness, in MM. TERRIER and REYMOND's book, *Chirurgie du cœur et du péricarde.* Paris, Félix Alcan, 1898. Price 3 fr.

Heat Fever and **Heat Shock.** *See* SUNSTROKE.

Hemiplegia.

Various Forms of	465	*Infantile Hemiplegia*	472
Condition at its onset	466	*Condition in long-standing Cases*	472

See also BRAIN—DISEASES OF VESSELS, PARALYSIS.

WHATEVER objection may be raised to the name hemiplegia it is so widely used, and so easily understood that no substitute, even were such forthcoming, is likely to displace it. By the term is understood a paralytic affection of the whole of one side of the body, with or without involvement of sensation, and clinical varieties of the ordinary condition depend upon the relative degree of the paralysis in the different parts of the affected side. Thus there is a condition in which the arm on the paralysed side is more affected than the leg; another in which the leg is more affected than the arm; another in which aphasia is superadded to the paralysis; another in which one half of each retina is also affected, giving rise to a condition of hemianopsia, and another in which the cranial nerves may be involved. These varieties depend upon the position in which the lesion causing the paralysis is situated.

Other varieties of hemiplegia are denoted by a descriptive title prefixed to them. Thus *crossed* hemiplegia (also called *alternate* hemiplegia) is a variety in which one side of the face (and perhaps also the external rectus muscle and also the sensation of one side of the face) is affected while the limbs on the *opposite* side are paralysed (see vol. ii. p. 60). *Double hemiplegia* (also known as *pseudo-bulbar paralysis*) describes a variety in which there is evidence of paralysis on each side of the body, the result of a lesion on each side of the cerebrum, or it may be of the pons, and the alternative name is used because of the close resemblance in the clinical characteristics of such cases to those of cases of degenerative affection of the nuclei of the bulb—cases of true bulbar paralysis. There is also a condition known as *hysterical* or *functional* hemiplegia, but the qualifying adjective does not imply any marked difference in the characters of the paralysis so much as in its originating conditions. The variety known as *infantile* hemiplegia, so far as the paralysis both in its extent and distribution is concerned, does not differ in any important essential from the adult type, but does differ in the time of onset, probably also in the nature of the lesion underlying it, and in having certain other symptoms associated with the paralysis.

In this article it is proposed to consider the condition of hemiplegia as it manifests itself clinically in an adult—(1) at the onset of the affection, (2) when the paralysis has become well defined in all its symptoms; and also the condition as it occurs in children—the so-called infantile hemiplegia just alluded to—differing in certain important particulars from the condition in the adult.

(1) *The Condition in a case of Hemiplegia at its onset.*—When the patient is seen the history of the mode of onset is of importance, not only as to the nature of the lesion but also in reference to prognosis and treatment. Frequently the account given is that the paralysis has occurred during sleep, although this is often difficult to verify. Not uncommonly the weakness has only become obvious when the patient gets out of bed, not aware of the paralysis, and then the history obtained is to the effect that the patient fell down and at once became paralysed, the true sequence of events being that the patient was already paralysed and fell down in consequence of the unilateral weakness. Such a patient when first seen is quite conscious and is paralysed on one or other side. The arm in the great majority of cases is more affected than the leg, and hangs usually limp and useless by the patient's side. In a case with such a history there is frequently no rigidity, and the deep reflexes may be normal at first or even diminished. In the paralysed limbs it is found that the most highly developed movements are those that have suffered most. Thus the fingers may be completely motionless while fair power can be exerted at the shoulder and elbow and even at the wrist. Occasionally it is found that the shoulder is much affected, and then the hand may not be completely paralysed, but even in such a case it is always found to be considerably impaired in its movements. In the lower limb the foot does not necessarily suffer most, and indeed the hip movements may be most interfered with.

In certain cases of hemiplegia, however, as already stated, the leg is more affected than the arm. This is due to a difference in the *position* of the lesion in the brain, not to any difference in its character,—the lesion being in such a case so situated as to affect more the leg centre in the cortex or the leg fibres in the white substance or in the internal capsule, than the corresponding structures for the upper limb. But on account of the fact that the leg is represented on each side of the brain—is bilaterally represented, to use the ordinary phrase—in a much greater degree than the arm, the paralysis of the leg, even in such a case as that now referred to, is never so complete as is the paralysis of the arm in an ordinary case. Thus it is rare to find in a case of this kind complete abolition of all movement in the leg, while it is not at all uncommon to find in a case in which the upper limb has suffered most that the arm is all but completely paralysed.

Besides the affection of the limbs that of the face may be obvious or may have to be carefully looked for. The lower part of the face suffers more than the upper, the reason being that the movements of the upper part of the face on the two sides are much more closely associated in their movements than those of the lower part of the face, and consequently they are represented in a correspondingly greater degree on the same side of the brain. Thus there may be, indeed there usually is, distinct failure of ability to raise the upper lip on the affected side in any attempt to show the teeth, while there may be no appreciable difference in the two sides when an attempt is made to wrinkle the forehead. The tongue is deflected to the paralysed side when protruded, the stronger muscles of the healthy side pushing it towards the weak side of the body.

The trunk muscles also suffer, but this is only seen, as a rule, on forced movement, the reason again being that the muscles of the one side are very closely associated in their habitual movements with those of the opposite side, and are consequently represented on each side of the brain, and therefore suffer comparatively little in a unilateral lesion. But a curious and important fact has been pointed out by Dr. Hughlings Jackson, viz., that in quiet respiration the movement of the paralysed side of the chest may be greater than that of the opposite side, while in voluntary respiration the side on which paralysis is present moves less than the other side.

Such, then, are the chief motor symptoms which are found in a case of this nature as it comes before the observer in an early stage, and the lesion causing such a condition may be situated in the cortex, underneath this, or in the internal capsule, crus, or pons. There may, however, be other conditions present. Thus marked impairment of sensation may coexist with the unilateral motor impairment—a condition pointing almost invariably to a lesion in the posterior part of the internal capsule where the sensory fibres from the whole of the opposite side of the body are transmitted to their still somewhat obscure termination in the cerebrum. In such a condition it is not unusual to find the leg more affected than the arm because of the closer proximity in this part, of the fibres subserving the leg movements to those subserving sensation. For a similar reason, when sensation is found to be impaired, the visual condition should be carefully examined, for as the fibres of the optic radiation run very near the spot at which such a lesion would exist, hemianopsia as a result of damage to them may, and frequently does, coexist with sensory impairment.

Such, then, is the usual condition as regards distribution and character of paralysis in a case of ordinary hemiplegia at the onset.

In a certain number of cases, however, the face, instead of being affected on the same side as the body, is paralysed on the opposite side. This is due to the fact that the lesion causing the paralysis is so situated as to affect the facial fibres *below* the nucleus, and therefore on the same side as that of the face to which they are distributed. Such a lesion, however, being situated *above* the decussation of the pyramids, will naturally cause paralysis of the limbs and body of the opposite side. This position of the lesion accounts not only for this so-called "crossed" paralysis, but it also accounts for certain features in the characters of the facial paralysis and for some associated paralysis. Thus, in such a lesion the upper part of the face is more affected than in cases of ordinary hemiplegia—the paralysis approaching closely in type to the ordinary type of facial paralysis resulting from an affection of the facial nerve (see "Facial Paralysis"). Then, also, in such cases there is usually associated with the facial paralysis an affection of the sixth nerve, causing paralysis of the external rectus muscle of the eye on the same side. The close proximity of the sixth nerve nucleus to the facial in the pons will explain this as well as an occasional involvement of the sensation of the face—from affection of the fifth nerve. Another form of crossed hemiplegia is also met with in which the functions of the third nerve are interfered with on one side and of the face and of the limbs on the opposite side. Such a condition can only result from a lesion—if there be a single lesion—in the *crus cerebri*, where the motor tract for the opposite side of the body and the third nerve of the same side are in close apposition.

A lesion also just above the decussation of the pyramids affecting the hypoglossal nerve for one side and the motor tracts of the other, may cause paralysis of the limbs on one side, and of the tongue on the opposite side, without any associated affection of the face. Such a lesion, however, is rare, and would almost certainly prove fatal on account of its proximity to vital structures in the bulb.

Reference has been made only so far to a condition of hemiplegia, commencing with little if any impairment of consciousness. Such a condition is usually the result of blocking of a small artery, although at first the dynamic effect of such a lesion may extend much beyond the area supplied by the occluded vessel, and so the initial paralysis be much more severe and extensive than it is ultimately. There may, however, in another class of cases be grave interference with consciousness at the onset, or even profound coma; and although all that has been said as to the character and distribution of the paralysis in a case of hemiplegia coming on without initial loss of consciousness is equally true of a case in which consciousness is lost at the onset, after consciousness has been restored, something must be said as to the initial condition in such a case, as it not infrequently renders the diagnosis considerably more difficult. The three most common causes of hemiplegia are—blocking of vessels by thrombosis, blocking by embolism, or the rupture of vessels. The conditions associated with the onset of the first have already been alluded to. Hemiplegia resulting from embolism is sudden in its onset. It may be unattended by loss of consciousness, and may very quickly pass off; or it may result in profound loss of consciousness lasting for hours at least, and convulsions may occur at the onset and be repeated during the unconsciousness. The character of the paralysis may be difficult to recognise during the unconsciousness. The whole condition of the patient may be one of apparently flaccid paralysis, but if careful watch is kept, the occasional voluntary movement of a limb on one side and the complete absence of any movement in those of the opposite side, may indicate the nature of the illness. Occasionally also there is considerable restlessness on the part of the patient, especially in cases of septic emboli, and on careful observation the absence of movement on one side may be detected. The knee-jerks may be completely lost, they will probably be so if the coma is profound, and deglutition may be interfered with as well as the action of the bladder and rectum. After the unconsciousness has passed off—although it must be remembered that it does not always do so—the condition of paralysis will be such as has already been described, and the position of the lesion will be determined from a consideration of the facts already mentioned.

A condition of cerebral hæmorrhage causing hemiplegia may have been preceded for some time by headache. On the other hand, it may occur suddenly when a patient is feeling particularly well. Consciousness may be lost at once, and a condition of profound coma supervene. Or the loss of consciousness may be slow, taking a few minutes or even, in the so-called "*ingravescent apoplexy*," a few hours. When consciousness is lost at once, and profound coma comes on, the hæmorrhage is probably into the ventricle, and it may be difficult to detect the signs of the unilaterality of the lesion.

If consciousness be slowly lost, the unilateral weakness will probably be distinct before the onset of unconsciousness, and when this comes on the one-sided nature of the paralysis may become somewhat masked. During unconsciousness marked deviation of the eyes may be observed, there is usually difficulty in swallowing, and there may be retention or incontinence of urine, and loss of control over the rectum. The skin usually assumes a greasy moistness, and trophic changes over points of pressure may occur rapidly. The unconsciousness may deepen to death, or there may be recovery, consciousness becoming restored after several days. When this happens there is not infrequently intense headache which is distressing and often intractable. The condition of paralysis is then recognisable, and its characters answer to those already described.

The *prognosis* in any case of hemiplegia in the early stages is beset with difficulties. In cases of embolic hemiplegia, if not very severe and occurring in young adults, it is often possible to predict a speedy recovery. But it must always be remembered that the condition causing the embolism if it still persist may give rise to a second one, sooner or later. The writer has known a patient three days after an attack of embolic hemiplegia from which he had recovered, have another attack from which recovery was only partial. And in many cases the recovery is but slight, and a condition of considerable paralysis remains permanently. Of cases of hæmorrhagic hemiplegia the prognosis as regards life is good after the first fortnight, and a certain degree of recovery can always be predicted. In the first fortnight, however, the patient is in imminent danger of death, either as a result of changes occurring around the first hæmorrhage, or because of the occurrence of a second. And as the vascular and other conditions underlying the hæmorrhage still persist, a second hæmorrhage in the near future is almost inevitable. In cases of thrombosis, due to senile atheroma, there is not, as a rule, an extension of the blocking, but recovery, partial it is true, but often considerable, takes place. The recovery in the first few days is often marked, probably, however, because much of the paralysis at first was dynamic in origin and not the result of actual structural change. After that recovery is slower, and is never complete. If the thrombosis occur in vessels, the seat of syphilitic endarteritis, or from blood states, such as are met with during pregnancy or the puerperium, complete recovery is not to be looked for. If the paralysis be the result of tumour or abscess, the degree of recovery depends upon the removability of the abscess or tumour by surgical or other means.

The pathological conditions underlying the paralysis and the differential diagnosis and treatment of these different conditions will be found described in the diseases of brain vessels (see "Brain," vol. ii.).

(ii.) It will now be well to consider the condition of hemiplegia as it is met with in a patient some months after the onset of the condition. Whatever may have been the nature of the initial lesion the subsequent condition is the same.

Motor Symptoms.—There is, in the ordinary case, weakness of the whole of one side of the body—the face, arm, and leg being most obviously affected, although the trunk muscles also suffer. It may just be mentioned here that although the more distinct weakness is of one side, the other side is also affected, but in a much slighter and less noticeable way. The lower part of the face suffers most, and there is an obliteration of the naso-labial fold and distinct drooping of the upper lip on an attempt at showing the teeth. The tongue is protruded towards the paralysed side. The arm is much less helpless than at the onset of the paralysis, but there is more movement at the shoulder and elbow than at the wrist or in the hand, although this is sometimes obscured by the presence of adhesions at the shoulder, considerably limiting movement there. The whole arm is stiff, or, to use the ordinary term, rigid, but this rigidity can at first be overcome, although later, through contractions taking place, structural alterations at the joints and in the muscles make even passive movements restricted and difficult. There is in the ordinary case much more affection of the finger movements than of any others. The leg is similarly affected, although in less degree. The rigidity, however, is distinct. In some cases of hemiplegia, as has already been said, the leg is more affected than the arm, and in such a case there is inability to walk for a long time even after a fair degree of power is present in the arm and even in the hand; yet in such a case, no doubt because of the bilateral representation of leg movement in the brain, recovery in the leg usually proceeds to such a stage as to permit walking.

The reflexes may be alluded to. In the leg there is usually ankle clonus and the knee jerk is exaggerated, and there may actually be a knee clonus. This exaggeration, although more marked on the paralysed side, is present also on the other, and even ankle clonus occasionally occurs on the so-called healthy side. In the arm there is exaggeration of the jerks elicited on tapping the wrist and elbow, and attention has recently been called by Babinsky and others to the fact that stroking the sole of the foot, in this and other conditions in which the lateral columns in the cord are degenerated, elicits a movement of extension of the big toe, the normal one being one of flexion. Sometimes by depressing the lower jaw and tapping the chin, a well-marked jaw jerk may be obtained.

Occasional irregular movements are present on the affected side in hemiplegia. The most common of these is athetosis, in which the hand on the affected side undergoes a cycle of slow involuntary movements. The movement may also affect the arm and shoulder, so that the arm is elevated above the head in a grotesque and striking manner. Similar movement may be present in the foot and at the ankle, rarely at the other parts of the leg, and not infrequently about the face and neck, the platysma being apparently the muscle most implicated. Marked tremor may also be present on the paralysed side, resembling that of disseminated sclerosis in being evoked only on movement, but both these forms of movement are much more common in the cases of hemiplegia occurring in early life, so-called *infantile hemiplegia,* to which reference will presently be made.

Besides these evidences of motor paralysis sensory impairment may also be present. This, as we have already said, is more likely to be met with in cases in which the leg is more affected, and consists in a blunting to all forms of sensory stimulation. The special senses may also be involved. Hemianopsia, *i.e.* abolition of function of the half of each retina on the side of the lesion, resulting in blindness of the half of each visual field on the paralysed side, is the form in which vision is affected. Hearing, taste, and smell may be impaired on the paralysed side.

This is probably the most suitable place in which to refer to the speech and articulatory defects in hemiplegia, because they are partly sensory and partly motor. In nearly every case even of *left* hemiplegia there is at first some difficulty with articulation resulting in a slurring or blurring of what are usually clearly articulated definite sounds. This may pass off in a few days, or weeks, but in a certain proportion of the cases persists throughout. In the majority of cases of *right* hemiplegia, however, speech proper is affected. The motor processes of speech may be interfered with, so that the patient is unable to fit words to things or to ideas, resulting in the condition known as *motor* aphasia. The patient knows what he wants to say, but cannot say it, yet is able to recognise it when it is said, and to understand anything spoken to him; or he may be unable to fit written characters to heard words, a condition known as *agraphia*. Another defect, however, may be present, so-called *sensory* aphasia, in which the patient is unable to understand what is said to him,—so-called auditory aphasia; or is unable to interpret seen objects, *e.g.* unable to read—so-called visual aphasia or word-blindness. All these defects only occur in cases of right hemiplegia, or in cases of left hemiplegia in a left-handed person. The writer has seen a case, however, of left hemiplegia with aphasia in a right-handed woman. This patient's father, curiously enough, was left-handed in a marked degree. And it should be remembered that all cases of right hemiplegia do not suffer from aphasia, and that those most likely to escape are for obvious reasons cases in which the leg suffers more than the arm (see APHASIA, vol. i.).

Trophic and other changes.—Besides the motor and sensory symptoms already described, certain changes, depending probably upon some change in the nutrition of the affected side, are usually present. Sometimes the limbs are colder and more blue, as if there were some venous engorgement due to vasomotor paralysis. Frequently there is a marked diminution in the size of the limbs as compared with those of the opposite side, the result in some degree, no doubt, of the absence of physiological exercise, in some degree, perhaps, of the actual cerebral lesion and its effect on nutrition. Often the diminution in the limb is associated with changes in the joints, especially the shoulder-joint, which may become fixed and immobile. When this is present the diminution in the size of the upper limb may be very striking.

A few words are also necessary in reference to mental changes which may be present. In the ordinary case these are not very marked, usually consisting in a certain degree of emotional instability, so that the patient easily laughs or cries, and in a tendency to be easily irritated. Occasionally in the early stage actual acute maniacal symptoms may supervene, but this is at least rare in the later stages, if it occurs at all.

In some cases convulsions occur at intervals after the paralysis is past. These may be unilateral in distribution, or bilateral. They probably are to be expected in cases in which the lesion is just underneath the cortex.

Actual neuritis may occur in a paralysed limb, characterised by great pain, glossy skin, and changed electrical reactions. Such a complication the writer has seen most frequently in people of a gouty constitution, especially if glycosuria happened to be present. It is nearly always associated with joint changes, especially in the shoulder-joint.

Treatment.—In the acute condition, at the time of onset, this must depend upon the diagnosis. If the hemiplegia is associated with a feeble pulse, if it has come on without loss of consciousness in an old person, the condition is probably one of thrombosis, and rest, quiet, easily assimilated food, and the somewhat free use of strychnia and alcohol, are the measures most likely to be successful. If the patient is younger and the condition is regarded as one due to syphilitic arteritis, then the usual antisyphilitic remedies must be energetically used. But when once thrombosis has occurred, and necrotic changes have taken place in the area subserved by the blocked vessel, even the most energetic treatment is not likely to be successful in preventing permanent paralysis. If the case be one of embolism, rest is essential. The cardiac condition must be carefully watched, and cardiac tonics used if necessary. If, however, unconsciousness be present, and the patient have a tense pulse and a hypertrophied heart, then probably intracranial hæmorrhage is the condition present, and free purgation and other means of lowering tension and ridding the blood of poisonous material are the beginning and the end of effective therapeutics. Should the hemiplegia be the result of tumour, abscess, or fracture, these conditions must be dealt with *secundum artem*.

As regards the condition when it has become chronic, not much in the way of treatment will influence it. Attention is to be chiefly directed to maintaining good nutrition and preventing contractions and other deformities. Contracture is nearly always flexor, and attention should be directed to securing adequate stimulation of the extensor muscles by means of electrical treatment or resistance exercises. Massage and passive movement are also useful in preventing or in minimising contractures.

The pathological changes underlying the condition described have already been dealt with in considering the results of disease of the brain vessels.

(iii.) *Infantile Hemiplegia.*—Although the state of a patient suffering from this form of paralysis, after the condition is well established, differs in no important particular from that first described, there are certain additional symptoms, especially associated with its onset, which mark it out as a distinct variety, worthy of special mention.

Infantile hemiplegia is the term applied to that form of hemiplegia which occurs in early life, usually in the first six years of life. It is ushered in, as a rule, with general malaise, with high fever and one severe unilateral convulsion or a series of such. The convulsion may spread so as to affect both sides. The condition of the child at this stage is one of grave and not infrequently fatal illness. The convulsive attacks may persist during several days, and when they cease the child is found to be paralysed on one side. At first the paralysis is flaccid, usually without sensory impairment, although hemianopsia is said to be frequently present. If the paralysis is right-sided, and the child had been able to speak, speech may be much interfered with, but only temporarily, the child probably regaining the power of expression by the education of the corresponding centres of the other hemisphere.

If a patient who has suffered in this way is seen a few years after the attack, the condition resembles closely that already described as occurring in the adult. There is the same spastic rigidity with contracture affecting the arm more than the leg, the same exaggeration of reflexes, and a similar but much more marked difference in size and development between the limbs on opposite sides, those on the paralysed side, especially the arm, being much smaller. The involuntary movements already alluded to, especially athetosis, is met with characteristically in this condition, and unilateral convulsions are of frequent occurrence. These may remain absent even for years after the initial convulsions, but they are apt to recur. In some cases the convulsions are severe, in others the attacks are more those of *petit mal*, and they may be associated with post-epileptic automatism. The mental condition is nearly always impaired, and not infrequently a condition of imbecility is present which renders the child suitable only for an asylum or a similar institution.

Pathology and Morbid Anatomy.—The condition is probably an inflammatory one of the cortex. Whether this is primarily in the cells or the result of vascular blocking, is not yet certain, but it seems not unlikely that an organised virus is the primary cause of whatever results in vessels or brain cells. When a child recovers from the initial grave condition and lives for years afterwards, the brain is then found to be in a cystic condition (porencephaly) with degeneration around it, a condition which, it is obvious, cannot inform us as to the primary cause of the brain lesion.

Prognosis and Treatment.—At the commencement of the illness life is gravely threatened, and death can only be prevented by careful and judicious treatment. Tepid sponging may do much to reduce the temperature; bromide by the rectum may control the convulsions, and should be given freely. A dose of calomel should be administered as soon as the condition is recognised, and care exercised that the child gets sufficient nourishment of a light, easily-digested quality.

If the acute condition be recovered from the prognosis is grave as regards anything like complete recovery of power, and the possibility of great physical impairment, of the frequent occurrence of convulsions, and of much mental change, must be borne in mind in forecasting the future. Not much can be done by drugs after the hemiplegia is established. The convulsions, however, can usually be completely controlled by the judicious use of bromide combined with arsenic and nux vomica, and by carefully chosen exercises and passive movements physical development may be aided and deformities prevented.

If the mental condition be impaired just after the onset there is not much chance that it will ever return to normal, and as soon as possible the child should be sent to an institution.

Hereditary Ataxia. *See* Paralysis.

Heredity.

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Heredity is a term for the relation of organic or genetic continuity which binds generation to generation. It is an anachronism to speak of it as a power, or principle, or force. Similarly, inheritance may be defined as all that the organism is or has to start with in virtue of its genetic relation to its parents and ancestors. The central problem of heredity is to arrive at an accurate conception of the genetic relation between successive generations; the central problem of inheritance is to measure the resemblances and differences in the hereditary characters of successive generations, and to find, if possible, some general formula which will sum up the facts.

Physical Basis of Inheritance.—If we mean by inheritance all that an organism is or has to start with in virtue of its genetic relation to its parents and ancestors, then it is plain that the physical basis is in the fertilised ovum. There is, as regards property, an obvious distinction between the inheritance and the person who inherits, but no such distinction is possible in biology, for the fertilised ovum *is* the inheritance and is at the same time the potential inheritor. This is a biological commonplace, as a statement of fact quite indisputable, nevertheless bristling with difficulties. Some of these difficulties, however, are incidental, not intrinsic. Thus, though it is interesting to ask how a heritable organisation, supposed to be very complex, may be imagined to find physical basis in a microscopic ovum and in a spermatozoon which may be only $\frac{1}{100000}$ of the ovum's size, the same sort of question may be raised in regard to ganglion cells; it is not distinctively a problem of heredity. It may, however, be recalled (1) that the physicists report that the image of a Great Eastern filled with framework as intricate as that of the daintiest watch does not exaggerate the possibilities of molecular complexity in a spermatozoon, whose actual size may be less than the smallest dot on the watch's face; (2) that in development one step conditions the next, and one structure often grows out of another, so that we are not forced to stock the microscopic germ-cells with more than initiatives; and (3) that development implies an interaction between the growing organism and a complex environment, without the stimulus of which the inheritance would remain unexpressed, and that the full-grown organism includes much that was not inherited at all, but has been acquired as the result of nurture or external influence. And, again, it is altogether inexpedient to lay on the shoulders of the student of heredity the burden of interpreting the orderly and correlated succession of events by which the fertilised egg-cell gives rise to an embryo. This is the unsolved problem of physiological embryology, and raises questions quite distinct from those of heredity and inheritance.

But when these incidental difficulties are set aside as irrelevant, there remains the intrinsic difficulty of accounting for the germ-cell's complex, ready-made organisation and marvellous potentiality. One suggestion is expressed in the theory of pangenesis, which occurred at intervals in the long period between Democritus and Darwin. On this theory the cells of the body are supposed to give off characteristic and representative gemmules; these are supposed to find their way to the reproductive elements, which thus come to contain representative samples of the various components of the body, and are therefore able to develop into an offspring like the parent. This theory involves many hypotheses, and is avowedly unverifiable in direct experience, but it is more to the point to notice that there is another theory of heredity which is on the whole simpler, which does, on the whole, fit the facts better.

This second theory is expressed in the phrase "germinal continuity," and has been independently expressed by a number of biologists, though Weismann has the credit of its elaboration. There is a sense, Mr. Galton says, in which the child is as old as the parent, for when the parent's body is developing from the fertilised ovum, a residue of unaltered germinal material is kept apart to form the future reproductive cells, one of which may become the starting-point of a child. In many cases scattered through the animal kingdom (*e.g.*, *Ascaris* and *Sagitta* among worms, *Moina* among crustaceans, *Chironomus* among insects, Phalangidæ among arachnids, *Micrometrus aggregatus* among fishes) the beginning of the lineage of germ-cells is demonstrable in very early stages before the differentiation of the body-cells has more than begun. Thus in the development of *Ascaris megalocephala* of the horse, according to Boveri, the very first cleavage divides the fertilised ovum into a cell which is the ancestor of all the somatic cells, and another, which is the ancestor of all the germ-cells.

But in many other cases, notably in plants and in the higher animals, the segregation of germ-cells is not demonstrable until a relatively late stage. Therefore, while the keystone of Weismann's theory is that the germinal material which starts an offspring owes its virtue to being materially continuous with the germinal material from which the parent or parents arose, he does not suppose a continuous lineage of recognisable germ-*cells* (for this is often unrecognisable), but a continuity of the germ-*plasm*; that is, of a specific substance of definite chemical and molecular structure which is the bearer of the hereditary qualities. According to Weismann, a part of the germ-plasm contained in the parent egg-cell is not used up in the construction of the body of the offspring, but is reserved unchanged for the formation of the germ-cells of the following generation. Thus the parent is rather the trustee of the germ-plasm than the producer of the child; and in a new sense the child is a chip of the old block. Similar material to start with, similar conditions in which to develop, *therefore*, like tends to beget like.

It should be carefully noticed that while early segregation of the germ-cells is in many cases an observable fact—and doubtless the list of such cases will be added to—the conception of the "germ-plasm" is hypothetical, just as the conception of *a* specific living stuff or "protoplasm" is hypothetical. In the complex microcosm of the cell we cannot point to any one stuff and say "this is protoplasm"; and it may well be that vital activity depends upon the interactions of several complex stuffs which, like the members of a carefully-constituted form, are characteristically powerful only in virtue of their inter-relations. Still less can we demonstrate the "germ-plasm," even if we were able to show that its physical basis is in the chromosomes of the nucleus. The theory has to be judged, like all conceptual formulæ, by its adequacy in fitting facts.

Dual Nature of Inheritance.—It is a familiar fact that, apart from exceptional cases (*e.g.* asexual multiplication and parthenogenesis), the inheritance of a multicellular organism is dual, part of it coming from the mother and part of it from the father. The more we know in regard to fertilisation, the clearer does the general fact become that there is an intimate and orderly union of maternal and paternal contributions. Professor E. B. Wilson sums up the present state of opinion somewhat as follows:—As the ovum is much the larger, it is believed to furnish the initial capital—including it may be a legacy of food-yolk—for the early development of the embryo. From both parents alike comes the inherited organisation which has its seat (according to many) in the chromosomes of the nuclei of ovum and spermatozoon. From the father comes the centrosome which organises the machinery of cleavage and distributes the dual inheritance equally between the daughter-cells. Recent discoveries confirm Huxley's prophecy (1878):—"It is conceivable, and indeed probable, that every part of the adult contains molecules derived both from the male and from the female parent; and that, regarded as a mass of molecules, the entire organism may be compared to a web of which the warp is derived from the female and the woof from the male." "What has since been gained," Wilson says, "is the knowledge that this web is to be sought in the chromatic substance of the nuclei, and that the centrosome is the weaver at the loom." Four saving clauses seem necessary at this stage in our discussion:—(1) What we have called the second great fact of inheritance does not imply that the dual nature of the inheritance must be *patent* in the full-grown offspring, for hereditary resemblance is often markedly unilateral. (2) Though inheritance is immediately dual, it is in quite as real a sense multiple, from ancestors through parents. (3) If Loeb is able to induce artificial parthenogenesis in sea-urchins' eggs exposed for a couple of hours to sea-water to which some magnesium chloride has been added; if

Delage is able to fertilise and to rear normal larvæ from *non*-nucleated ovum-fragments of sea-urchin, worm (*Lanice*), and mollusc (*Dentalium*), we should be chary of accepting too readily the conclusion that the nuclei are the exclusive bearers of the hereditary qualities. (4) The fact that an ovum without any sperm-nucleus, and an ovum-fragment without any but a sperm-nucleus, can in some cases develop into a normal larva, points to the conclusion, which other facts also suggest, that each germ-cell, whether ovum or spermatozoon, bears a complete equipment of hereditary qualities.

Different Degrees of Hereditary Resemblance.—The big treatise of Prosper Lucas (1847) may be said to close the period of *proving* hereditary resemblance. It is now legitimately taken for granted that the present is the child of the past, and that the past is represented in the child. An organism's start in life is vigorously determined by its parents and ancestors; not only specific characters, but trivial idiosyncrasies—not only physical qualities, but mental as well—not only the normal, but the abnormal may be transmitted. At the same time, it should be noted that this department of the study of heredity is by no means closed; thus some morbid conditions are much more likely to be transmitted than others, and we ought to have statistical estimates of the probabilities of transmission in each case. And, again, there are some subtle qualities whose heritability must not be assumed without evidence. Thus it is of great importance that Karl Pearson has recently supplied, for certain cases, definite proof of the heritability of fecundity, fertility, and longevity.

The large fact of inheritance which confronts us is the sensible stability of type from generation to generation. It is summed up in the familiar saying, "Like begets like." We know, however, that this saying is insufficient, since variation is as striking a fact as complete hereditary resemblance. A variation may imply some incompleteness in the offspring's re-expression of the parents' hereditary qualities, or it may imply the appearance of something new,—some novel molecular arrangement in the germ-plasm. In any case it leads us to modify the familiar saying, and to state more cautiously that like *tends* to beget like. But this platitude does not sum up even our familiar experience, and thus we are led to consider the different degrees of hereditary resemblance, for which a confused classification and a troublesome terminology has been suggested. The three most important cases seem to be blended, exclusive, and particulate inheritance. (*a*) In *blended* inheritance, the characters of the two parents, *e.g.* in regard to a particular feature, such as the colour of the hair, are ultimately combined in the offspring. This is particularly well seen in some hybrids, and is probably the most frequent mode of inheritance. (*b*) In *exclusive* inheritance, the expression of maternal or of paternal characters in relation to a given feature, such as eye colour, is suppressed. The resemblance is unilateral, and often crossed, the son taking after the mother and the daughter after the father. (*c*) In *particulate* inheritance there is in the expression of a given character a part which is wholly paternal and a part which is wholly maternal. Thus an English sheep-dog may have a paternal eye on one side, and a maternal eye on the other. Suppose the parents of a foal to be markedly light and dark; if the foal is light-brown or gray the inheritance is blended; if light or dark it is exclusive; if piebald, it is particulate in its mode of inheritance for that feature.

As already hinted, the different modes of inheritance are often well illustrated in hybrids between different species or breeds. The hybrid may be thoroughly intermediate between its parents, the blending being more like the mingling of two pigments than the interweaving of warp and woof.

Or it may show an exaggeration of the characters of one parent, often with little apparent realisation of the peculiarities of the other. These two cases correspond to blended and exclusive inheritance in ordinary mating within the same breed. But the hybrid may in other cases be very different from either parent, and exhibit features which appear to be novel, or seem interpretable as the re-assertion of the characteristics of a remoter ancestor. In short, it may show either a new variation or a reversion. Perhaps the most extraordinary fact is that at least two of these different modes of inheritance may be illustrated in one brood or litter of hybrids.

From another point of view we may express the facts in terms of the quality of prepotency. It seems certain that in respect to certain characters the paternal inheritance is often more potent—more capable of finding expression—than the maternal, or *vice versa;* thus in man the father tends to be prepotent in the matter of stature, and breeders give many instances where certain, even trivial, characters of the sire or the dam reappear persistently in the offspring irrespective of the nature of the other parent. If, as Ewart and others maintain, this quality of prepotency tends to be developed by inbreeding, it may be frequent in nature, especially among gregarious and isolated groups, and it may explain the persistence of new variations in their incipient stages. It is interesting to note Reibmayr's thesis that the evolution of a successful human race implies alternating periods of dominant inbreeding and dominant cross-breeding. The former gives fixity to character, the latter averts degeneracy, and stimulates those new variations which form the raw material of progress.

Until more precise data accumulate in regard to blended, exclusive, and particulate inheritance, it will not be possible to simplify the matter with any security, but attention may be directed to Weismann's theoretical suggestion of a germinal struggle in the arcana of the germ-cells, a struggle in which the maternal and paternal contributions may blend and harmonise, or may neutralise one another, or in which one may conquer the other, or in which both may persist without combining.

Finally, in this connection, we must note that while it is a matter of observation that there are great differences in the degree in which offspring resemble their parents, it is a matter of conjecture that lack of resemblance must be due to incompleteness in the inheritance. Indeed, the fact that the resemblance so often reappears in the third generation makes it probable that the incompleteness is not in the inheritance, but simply in the expression of it. The characters which seem to be absent, to "skip a generation" as we say, are probably part of the inheritance all the time, but they remain latent, being neutralised, silenced (we can only use metaphors) by other characters, or unexpressed because of the absence of the appropriate stimulus. A neglect of this distinction is a frequent source of misunderstanding.

Filial Regression.—From generation to generation there is a tendency to keep up a specific average. "The large," Galton says, "do not always beget the large, nor the small the small; but yet the observed proportion between the large and the small, in each degree of size and in every quality, hardly varies from one generation to another." This is partly due to natural elimination, weeding out the extraordinary and the abnormal, often at or even before birth. But it is to be primarily accounted for by what Galton calls "filial regression." Karl Pearson gives a clear illustration:—take fathers of stature 72 inches, the mean height of their sons is 70·8,—a regression towards the mean of the general population; on the other hand, fathers with a mean height of 66 inches give a group of sons of mean height 68·3 inches,—again nearer the mean. "The father with a great excess of the character contributes sons with an excess, but a less excess of it; the father with a great defect of the character contributes sons with a defect, but less of it."

As Galton puts it, human society moves as a vast fraternity. The sustaining of the specific average is not due to each individual leaving his like behind him; it is due to a regression which tends to bring the offspring of extraordinary parents nearer the average of the stock. In other words, children tend to differ less from mediocrity than their parents. This big average fact is to be accounted for in terms of that genetic continuity which makes an inheritance not dual but multiple. A man is the product not only of his parents, but of his ancestry, and "unless very careful selection has taken place, the mean of that ancestry is probably not far from that of the general population." Pearson continues, "It is the heavy weight of this mediocre ancestry which causes the son of an exceptional father to regress towards the general population mean; it is the balance of this sturdy commonplaceness which enables the son of a degenerate father to escape the whole burden of the parental ill."

Law of Ancestral Inheritance.—Perhaps the most important general conclusion which has yet been reached in regard to inheritance is that formulated in Galton's law of ancestral inheritance, to which this authority was led by his studies on the inheritance of human faculties, and more particularly by a series of studies on Basset hounds. According to Galton's law, "the two parents between them contribute *on the average* one-half of each inherited faculty, each of them contributing one quarter of it. The four grandparents contribute between them one quarter, or each of them one-sixteenth; and so on, the sum of the series $\frac{1}{2} + \frac{1}{4} + \frac{1}{8} + \frac{1}{16} +$ etc., being equal to 1, as it should be. It is a property of this infinite series that each term is equal to the sum of all those that follow, thus: $\frac{1}{2} = \frac{1}{4} + \frac{1}{8} + \frac{1}{16} +$ etc.; $\frac{1}{4} = \frac{1}{8} + \frac{1}{16} +$ etc., and so on. The prepotencies or subpotencies of particular ancestors, in any given pedigree, are eliminated by a law which deals only with *average* contributions, and the varying prepotencies of sex in respect to different qualities are presumably eliminated."

This law of ancestral inheritance, which states that each parent contributes on an average one-quarter, each grandparent one-sixteenth, and so on, must not be accepted as a dogmatic conclusion, but as an approximate statistical formula, and it must be noted that it applies most convincingly to cases of *blended* (not exclusive) inheritance. Pearson's paper on the "Law of Reversion" (1900) should be read as a supplement to Galton's *Natural Inheritance.* In connection with the number of ancestors and the mosaic or multiple nature of inheritance, it may be useful to recall that intermarriages greatly reduce the theoretical number of ancestors. Thus while Kaiser Wilhelm II. might have had 16, 32, 64, 128, 256, 512, 1024, 2048, 4096 ancestors in generations iv.-xii. respectively, he actually had 14, 24, 44, 74 in generations iv.-vii., and probably 116, 177, 256, 342, and 533 in generations viii.-xii.

Reversion.—This term may be conveniently used to include cases where, *through inheritance*, an individual exhibits some character or characters not expressed in the parents, but known to occur in ancestors. The character, normal or abnormal, whose reappearance is called a reversion, may be found within the verifiable family, within the breed, within the species, or even in a presumed ancestral species. Karl Pearson defines a *reversion* as "the full appearance in an individual of a character which is recorded to have occurred in a definite ancestor of the same race," while *atavism* is "a return of an individual to a character not typical of the race at all, but found in allied races supposed to be related to the evolutionary ancestry of the given race." But as the two words have been used by some others in the converse way, or as equivalent, and as it seems only a distinction of degree, the single term reversion may here suffice.

Good illustrations of reversion are furnished by hybrids. Thus, in one of Ewart's experiments a pure white fantail cock-pigeon, of old-established breed, which in colour had proved itself prepotent over a blue pouter, was mated with a cross previously made between an owl and an archangel, which was far more of an owl than an archangel. The result was a couple of fantail-owl-archangel crosses—one resembling the Shetland rock-pigeon, and the other the blue rock of India. Not only in colour, but in shape, attitude, and movements there was an almost complete reversion to the form which is believed to be ancestral to all the domestic pigeons. The only marked difference was a slight arching of the tail, which was, however, 12-feathered as usual, in contrast to the 30-feathered one in the fantail.

But great care is necessary in arguing from the results of hybridisation to those of ordinary mating, and even if some of the phenomena of exclusive inheritance seem to demonstrate reversion to a near ancestor, we need a broader basis of facts than we have at present before we can generalise. Karl Pearson (1900) has recently sought to formulate a general law of reversion supplementary to Galton's law of ancestral inheritance.

Many phenomena have been labelled reversions on the flimsiest evidence. Thus the occurrence of a Cyclopean human monster with a median eye has been called a reversion to the ascidian, and gout has been called a reversion to the reptilian condition of liver and kidneys. Often there is not the slightest attempt to eliminate the phenomena of arrested development or of abnormalities induced from without. Often, too, there has been no scruple in naming or even inventing the ancestor, to whom the reversion is supposed to point, although definite evidence of the pedigree is awanting; and the vicious circle is not unknown of arguing to the supposed ancestor from the supposed reversion, and then justifying the term reversion from its resemblance to the supposed ancestor. Little allowance has been made for coincidence, and the postulate of characters remaining latent for millions of years is made as glibly as if it were just as conceivable as a throwback to a great-grandfather.

Reversion is a phenomenon of exclusive inheritance, and the theoretical conception which it implies is that characters may be latent for a generation or for generations, or, in other words, that certain potentialities or initiatives which form part of the heritage may remain unexpressed for lack of the appropriate liberating stimulus, or for other reasons, or may have their normal expression disguised. There does not seem to be anything in this conception which is at variance with more securely established generalisations. But the danger is in applying the interpretation. Even when an individual exhibits the reappearance of an ancestral character which was not in his parents, this is not necessarily due to the reassertion of latent elements in the inheritance. It may be a case of ordinary regression; it may be a case of arrested development; it may be an individually acquired modification adventitiously induced, apart from inheritance, by a recurrence of suitable conditions of function or environment; it may be an extreme variation whose resemblance to an ancestral characteristic is a coincidence; and so on. In short, what are called reversions are probably in many cases misinterpretations.

Transmission of Acquired Characters.—The question of the transmissibility of acquired characters has been much discussed within recent years, but there has not always been a clear apprehension of the point at issue. The individuals composing a species are neither quite like one another nor quite like their parents, and it is possible to measure these "*observed differences.*" As we come to analyse them, we discern that many structural peculiarities of the body can be shown by experiment to be definitely related to some alteration or peculiarity in environment or in function, that these are not even hinted at in the young forms, but begin to appear when the particular conditions begin to operate. These are called by biologists "*modifications*" or "*acquired characters,*" and it is in regard to these and these alone that the real argument has concerned itself. They may be defined as structural changes in the body of the organism induced by changes in the environment or in the function, and such that they transcend the limits of organic elasticity and therefore persist. Now when we eliminate from the total of observed differences of structure the somatic modifications which we have detected, there remain a number of differences which we call "*variations.*" These cannot be shown to result directly from functional or environmental stimuli operating upon the body; they are often hinted at even before birth; and they are not alike even among similar forms whose conditions of life seem absolutely uniform. Little that is certain is known in regard to their origin, but it is supposed that they result from changes in the germinal material before or in fertilisation; they are therefore called germinal, blastogenic, or congenital variations, and their transmissibility is indubitable. The precise question is, therefore, whether the modifications of the body can so specifically affect the reproductive cells that the next generation will inherit, in some measure at least, the modification acquired by the parent or parents. In other words, may the results of "nurture" be transmitted, or is it the "nature" alone that constitutes the inheritance?

Some of the frequent misunderstandings of the question must be referred to to clear the ground. (1) There is no relevancy in citing cases where a particular somatic change appears generation after generation, *e.g.* shortsightedness or gout, unless it is clearly shown that the change in question is really a "somatic modification," and not a congenital variation whose transmissibility is admitted by all. (2) It is a misunderstanding to cite cases where unicellular organisms, such as bacteria or monads, have been profoundly modified by culture, so that, for instance, the descendants of a virulent microbe are gradually led to lose their evil potency. This is irrelevant, because in regard to unicellular organisms we cannot draw the distinction between germinal matter and soma on which the definition of an acquired character depends. (3) There is little relevancy in citing cases where a particular bodily change appears generation after generation, unless it can be shown that the change reappears *in virtue of inheritance,* and not simply because the conditions of function and environment which evoked it in the first instance are still persisting to evoke it in those that follow. Reappearance is often confused with inheritance. (4) It is necessary to distinguish between the possible inheritance of a particular modification and the possible inheritance of indirect results of that modification. It is likely that some important modifications influence the general vigour of the body, and thus through nutrition the reproductive cells; but unless the offspring change *in the same direction* as that exhibited in the original parental modification, we are not warranted in speaking of the inheritance of an acquired character. (5) It is apt to be fallacious to appeal to data from not more than two generations. It has often been pointed out that mammals, such as sheep, taken to a new country, exhibit a change in the character and length of the hair, and that their progeny exhibit the modification in a still more marked degree. But unless statistics of the third generation at least are presented, such cases are of no value, for it is only natural that the second generation should show the modification in a more marked degree than their parents did, since the offspring were subjected to the modifying influence from birth, whereas their parents were influenced only from the date of their importation. (6) It is necessary to appreciate the distinction between a change of the reproductive cells *along with* the body, and a change of the reproductive cells *resulting from and representative of* a change in the body. Some poisoning of the system on the parent's part by alcohol, opium, or some virus, may be followed by degeneracy in the offspring. But if the fact be admitted, what is the correct interpretation? In some cases what is really inherited may be the degeneracy of nature which led the parent to, say, alcoholism, and which finds the same or another expression in the child. In other cases it may be that what looks like inheritance is the result of early infection before or soon after birth. In other cases it may be that there was in the parent a poisoning of the whole system—reproductive cells as well as body—which is not a case by which to test the transmissibility of an acquired character. (7) There is no doubt a sense in which every acquired character is congenital, for there must have been in the nature of the organism the rudimentary possibilities of it; and there is a sense in which every congenital character may be said to be acquired, since it needs to be nurtured by appropriate conditions if it is to develop. And yet the distinction is not a verbalism. For although we do not suppose that the environment is creative, and although we must admit that the potentiality of the acquired character must be in the nature of the organism, just as the possibility of an explosion in the barrel of gunpowder, yet, as a matter of fact, it is possible to distinguish between the actual modification which we see and measure and the possibility of it which we presuppose. Similarly, while it is very true that the potentialities so marvellously embodied in the fertilised ovum require appropriate environing conditions if they are to be realised, for, as His observed long ago, " it is a piece of unscientific mysticism to suppose that heredity will build up an organism without mechanical means," yet this does not affect the validity of our definition of an acquired modification as distinguished from a congenital specific character.

The main argument against a belief in the transmissibility of acquired characters is simply that the evidence for the affirmative is extremely unsatisfactory. But this position is corroborated if we accept the view that the germ-plasm or the material basis of inheritance is in a marked degree apart from the general life of the body, and sometimes segregated at a very early stage in development. For this view raises a presumption against the likelihood of the germ-plasm being readily affected in a specific and representative manner by changes in the nature of the body cells. It must be allowed, however, that our inability to conceive of the mechanism by which an acquired character of the brain may affect the reproductive cells in a specific and representative manner does not prove that the supposed influence is an impossibility.

The argument in favour of the view that acquired characters may be transmitted is found in the large number of facts which may be readily interpreted on this hypothesis, and on a few facts which seem directly to suggest it. And the affirmative position is strengthened by a consideration of the unity of the organism. In many plants the distinction between somatic cells and germ-cells can hardly be drawn, and even if we keep to animals the bonds between the body and its germ-cells are often very close. The blood and lymph or other body fluids form a common medium for the various parts of the organism. Alteration of diet in the early youth of some animals, such as tadpoles, may determine the predominance of one sex or the other through influences which must pass from soma to germ-cells. Various poisons may affect the bodily system and the reproductive organs at the same time, and there are real though dimly understood correlations between the gonads and the rest of the system. It is therefore erroneous to think of the germ-plasm as if it led a charmed life uninfluenced by any of the accidents and incidents in the daily life of the body which bears it. No one believes this, Weismann least of all, for he finds one of the chief sources of congenital or blastogenic variation in the nutritive stimuli exerted on the germ-plasm by the varying state of the body. But it is one thing to admit that the germ-plasm has no charmed life nor insulation from bodily influences, and quite another thing to believe that a change in the body, induced by use or disuse or by change in surroundings, can influence the germ-plasm in such a definite way that the offspring will exhibit the same modification which the parent acquired, or even a tendency towards it.

Of the direct evidence suggestive of the transmissibility of acquired characters a few representative samples may be given. (1) The Panjabis are said to show peculiarities of musculature and skeleton, which are related to the frequency with which these people assume the squatting posture. But it may be that these modifications are acquired during each individual lifetime. The evidence is inconclusive, and we may set against it the case of the compressed foot of Chinese ladies, for there seems to be no evidence that the long-continued deformation has resulted in any hereditary change in the Chinese baby's foot. (2) The alleged dwindling of the little toe is instanced as a case of the inheritance of a modification induced by tight boots. But the evidence is flimsy; a dwindling has also been alleged in savages who do not wear boots; it is possible that there is in man, as there was in the horse-stock, a congenital variation in favour of a reduction of digits; and there are other possible interpretations. (3) In 1796 the utmost speed of the English trotter was a mile in 2 minutes 57 seconds; decade after decade the speed and the percentage of swift trotters increased; finally, there has been evolved a breed who can trot a mile in 2 minutes 10 seconds. This has been claimed as evidence of the cumulative transmission of the results of exercise or nurture. But this interpretation overlooks the results of selective breeding which may have increased the congenital swiftness, and the process of elimination which persistently weeded out the less swift from the stud. (4) In 1875 Schmankewitsch was able to transform one type of brine-shrimp, *Artemia salina*, in the course of generations into another type, *Artemia milhausenii*, by lessening the salinity of the water; and conversely, by increasing it. The results are open to criticism on several grounds, but it is enough to recognise that Schmankewitsch experimented with a *progressively changing environment* on a series of generations, and that the result is interpretable as due to modifications hammered on each successive generation without there being any inheritance of these modifications. It is also possible that the reproductive cells before or after liberation were directly affected by the continuous change of salinity. (5) The fact that negroes and Mongolians are relatively immune to yellow fever has been cited as proof positive of the inheritance of an acquired character. But it may be that the quality of immunity was originally a congenital peculiarity, which has become dominant in the race by the elimination of those who were not immune. If it be objected that there are cases where a mother rabbit or guinea-pig has been artificially rendered immune to certain diseases, and has had young ones born immune, it may be answered that this is probably due to a kind of infection before birth, some antitoxin having passed from the mother to the unborn young. (6) The case of supposed modification-inheritance which has attracted most attention is that of Brown-Séquard's guinea-pigs. In a series of experiments extending over many years (1869-1891), Brown-Séquard showed that a partial section of the spinal cord, or a section of the sciatic nerve, was followed after some weeks by a peculiar morbid state resembling epilepsy. The offspring of the animals operated on were frequently decrepit, and a certain number showed a tendency to the so-called epilepsy.

As the original state may also be induced by bruising the sciatic nerve without cutting the skin, or by striking the animals on the head with a hammer, it seems unnecessary to consider the suggestion that the influence on the offspring was due to microbic infection. As a similar state may be induced in the dog by injury to the cerebral cortex, and was said in this case also to reappear in the offspring, it seems that we have not to deal with a peculiarity of the guinea-pig alone. As the epileptic state does not occur spontaneously in guinea-pigs, we may exclude the possibility of coincidence and the suggestion that captive guinea-pigs are nervously morbid. As the tendency to epileptic fits (which did not last long) was seen only in the offspring of animals which had been operated upon, and was only manifested after appropriate stimulus, especially after irritating an "epileptogenic" zone behind the ear on the same side as the original injury, we may exclude the suggestion that the epileptic tendency was imitative. Brown-Séquard's results have been confirmed by Dupuy (1890), Westphal (1871), Obersteiner (1875), and Romanes (1895); and no contrary evidence has as yet set aside the suggestion of modification inheritance which the case conveys.

On the other hand, it should be noted that the results are very various. In one set of experiments (Obersteiner, 1875), out of thirty-two young ones born of artificially epileptic parents, only two showed symptoms of epilepsy. The results in Brown-Séquard's cases were very various,—general feebleness, motor paralysis of the limbs, trophic paralysis resulting in loss of toes, cornea, etc., and only in some cases epilepsy or some similar nervous disorder. It seems fair to say that what was inherited was decrepitude; the modification was too violent to be a fair case; it disturbed the whole organism, nutritive and reproductive functions alike, and was thus almost bound to cause abnormality in the offspring.

Although the evidence suggesting the transmissibility of acquired characters seems altogether inconclusive, it may be noted that somatic modifications have enormous individual importance. If they are not transmissible, the importance of securing good "nurture"—both functional and environmental—is rather increased than diminished. It has also been pointed out by several naturalists, that although modifications may not be of direct value in evolution (if they are not transmissible), they may be of great indirect value by acting as the fostering nurses or shields of congenital variations in the same direction. Thus, if we suppose swarthiness to become a condition of survival in a given country, there would probably be some inhabitants with a strong natural or congenital tendency in this direction; there would probably be others in whom the congenital variation towards swarthiness was weak and incipient; there would probably be others who had simply a susceptibility to acquired swarthiness; and there would probably be others who were persistently blonde. The first would in the course of natural selection tend to become in themselves and in their progeny the dominant stock; the last would tend to be rapidly eliminated; but it is conceivable that those who made up for their lack of natural swarthiness by a great susceptibility to acquired swarthiness would also be very successful; in other words, it is conceivable that the modification, though never taking heritable root, would serve as a life-saving screen until coincident congenital variations in the direction of swarthiness had time to grow strong. The case is hypothetical, but the idea is applicable to realities.

Telegony.—The term telegony has been applied to the doubtful, certainly rare, but if true very remarkable occurrence of cases where an offspring resembles not so much its father as a previous mate of its mother. In other words, telegony is the supposed influence that a male may, through effective impregnation of a female, exert on offspring subsequently borne by the same female to a different sire. To take a simple instance, the race-horse "Blair-Athol" had a very characteristic blaze or white bald face; and it is said that mares which had borne foals to "Blair-Athol" subsequently produced to quite different stallions foals with the "Blair-Athol" blaze. The alleged cases are of much interest, but many of them are ill-authenticated, and others are capable of being differently interpreted. At the same time some explanation must be found for the fact that a belief in telegony is widespread among practical breeders.

Telegony has been alleged to occur in man, horses, cattle, sheep, pigs, dogs, etc., and even in pigeons, but even the most circumstantially recorded cases, such as Lord Morton's Arab mare, are far from satisfactory, and the majority cannot be called scientific data at all. In his "Penycuik Experiments" Cossar Ewart (1899) proved this at least, that telegony does not generally occur even when favourable conditions are afforded; only in a very small percentage of cases was there anything even suggestive of telegony. Moreover, where peculiar phenomena of inheritance were observed they seemed to be readily explicable on the "reversion hypothesis." The general nature of the experiments may be illustrated by one of the best cases. A Rum pony mare "Mulatto," of remarkably pure breed, was served by a Burchell zebra stallion "Matopa," and the result (in August 1896) was "Romulus," whose markings were quite different from those of his sire, being suggestive rather of the Somaliland zebra. In 1897 "Mulatto" had a bay colt foal to a gray Arab stallion, and this foal—unfortunately short-lived—gave no evidence of telegony. The stripes which most frequently occur in horses were absent; there were others which are not uncommon in horses; but the most distinct markings (not that any were strongly developed), namely, those across the croup, were of a sort extremely rare both in foals and horses. In short, the marking of "Mulatto's" second foal was puzzling, but in no definite way suggestive of the influence of the previous zebra sire. In this, as in other cases, the verdict as to the occurrence of telegony was non-proven. It is evident, however, that *if* telegony be a reality which occurs very rarely and under peculiar conditions, the probabilities are many against an experimenter realising these conditions in a short time; and therefore it is interesting to notice that Karl Pearson, working by statistical methods, was unable to find any quantitative evidence of a steady telegonic influence in man.

Till we are sure of the facts to be explained, the discussion of interpretations is gratuitous, but a brief summary may be given. (*a*) It has been suggested that the phenomena are simply illustrations of reversion, and that the resemblance between the offspring and a previous sire with which it has no genetic relation is a coincidence. The plausibility of this explanation will vary in different cases. Thus, Finn points out that the occurrence of feather-legged fowls from pure Dorking parents, or of polled lambs from black-faced horned parents, cannot be set down to reversion, "feather-legged fowls and polled sheep not being ancestral types." All depends, however on the proved purity of the parent breed. (*b*) Another theory interprets telegony as due to maternal impression, the supposition being that the mental image, etc., produced in the mother by the first sire exerts an influence on subsequent germs, or on their development by another sire. But see "MATERNAL IMPRESSIONS." (*c*) Weismann and others have suggested that spermatozoa of the first sire may reach the ovary, and become associated with immature ova, or may be in some way stored; and that a belated fertilisation may coincide in time with a second coitus by a different sire, to which the offspring would be naturally referred. Were this the case, we should expect to find cases where offspring were produced without any second sire at all, and no such cases (among higher animals at least) are known. (*d*) Somewhat subtler is the suggestion—often called the "infection hypothesis"—that the seminal matter of the first sire may, apart from the fertilisation of an ovum or ova, influence the reproductive organs or the constitution of the mother in such a way that subsequent gestations (following impregnation by another sire) may be affected. The probability of some physiological influence is probable, but it is difficult to conceive that the influence should be of so precise a nature as to evoke in offspring by a second sire a resemblance of the first. (*e*) Perhaps the most plausible theory is, that the mother is influenced through the fœtus during pregnancy, and that the influence reacts on subsequent offspring. This so-called "saturation hypothesis" suggests that some of the characteristics of the sire, while expressing themselves in the development of the embryo, may, as it were, saturate into the dam, and affect her constitution in such a precise way that her offspring by subsequent sires may through maternal influence be affected with some of the characteristics of the first. Thus, Sir William Turner (1889), in discussing Lord Morton's case, says: "I believe that the mother had acquired during her long gestation with the hybrid the power of transmitting quagga-like characters from it, owing to the interchange of material which had taken place between them in connection with the nutrition of the young one. . . . In this way the germ-plasm of the mother, belonging to ova which had not yet matured, had become modified whilst still lodged in the ovary. This acquired modification had influenced her future offspring derived from that germ-plasm, so that they in turn, though in more diluted form, exhibited zebra-like markings." It is conceivable that something like this may occur in the case of a poison or protective antitoxin, which might diffuse in and out. We can imagine that a sire infected with some virulent disease, and showing certain structural disturbances associated therewith, may have offspring which are similarly affected, and that the influence from them may pass before their birth into the constitution of the mother, and so affect her that subsequent offspring by a healthy sire are diseased after the manner of the first. But while we have some facts to go upon in regard to the diffusion of toxins and antitoxins, we have none as yet which warrant us in supposing the diffusion of structural characteristics or of the germinal representatives of these.

It remains to ask for some explanation of the widespread belief in the occurrence of a phenomenon, the scientific evidence for which seems so slender. There is no doubt, we are told, that the value of a pure-bred bitch at once goes down if she has been accidentally lined by a mongrel, and it is possible that there may be good reason for this, apart from the fact that the episode is not one which figures well in the books. It is possible that the constitution and temper of the bitch may be subtly affected by a coitus—especially fertile coitus—with a dog of inferior strain, and that the deteriorated constitution may react upon future offspring although real telegony does not ensue. It is hardly sufficient to remind ourselves that people are indescribably careless about their scientific beliefs, and that breeders are often too superstitious, for considerations of money value have a potent effect in evolving carefulness, and breeding is gradually becoming an art based on scientific conclusions. There must be some basis for the widespread belief, and the answer given by the practical men themselves is that they have abundant experience of telegony. This leads us to look for phenomena which might be readily mistaken as telegonic, and there can be little doubt that Ewart is right in thinking that the mistake is in the misinterpretation of *reversions*. The unexpectedness of results when different races are crossed is well known. A dark bantam hen, paired with an Indian game Dorking, produced, amongst others, a cockerel almost identical with a jungle fowl (*Gallus bankiva*), that is with the original wild stock. What occurs when different races or breeds are crossed may occur on a smaller scale when individuals of the same breed, but of different strains, are crossed. Reversionary phenomena, however they may be theoretically interpreted, are frequent, and when they occur they are to the practical breeder usually disappointing. In search for an interpretation, he sometimes thinks that he finds one in telegony; that is to say, he gives the blame of the reversionary phenomenon not to the immediately preceding crossing, which may have been theoretically correct, and should have turned out well, but to some remoter, less careful, or perhaps accidental crossing. In this way the remoter sire is made the scape-goat for the reversion, and the belief in telegony has grown.

Inheritance of Disease. — The study of the heritability of diseased states has not disclosed any general fact which is not observable in normal cases; and it is only for convenience that the two aspects should be separated. Since Lucas (1847) collected his data showing the heritability of malformations, numerical abnormalities, and many diseased states of body and mind, a more critical study has led physicians to formulate a number of distinctions between real and apparent inheritance. As these make for progress they may be briefly illustrated. For notes on the heritability of particular diseases the separate articles should be consulted.

(*a*) The reappearance of a diseased condition in successive generations does not prove that it has been transmitted, or that it is even transmissible. The Alpine plants which Nägeli brought to the botanical garden at Munich became so much changed that they were hardly recognisable as the same species, and their descendants were likewise transformed. There was no doubt as to the reappearance of the unusual characters, but there was every doubt that the reappearance was due to inheritance. That it was due to the persistence of the new conditions, and to the changes which these directly impressed on each successive crop, was shown by the fact that when the plants were removed to poor, gravelly soil, the southern modifications disappeared, and the plants were retransformed into their original Alpine state. So it is with many diseased states which reappear generation after generation, not because they have been transmitted, but because of the persistence of the unhealthy stimuli in function or in environment which originally evoked them.

(*b*) Even when a child is born with symptoms or definite expressions of a disease, it does not follow that the disease was part of the inheritance.

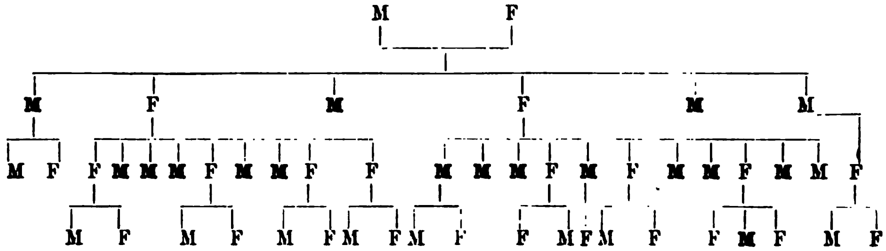
It may have been acquired by infection through the mother during the fœtal period. This may be illustrated in some cases of syphilis. Similarly, there is a growing body of evidence to show that in certain mammals (if not also in man) there may be a passage of antitoxin substances from the blood of an artificially immune mother to the blood of the fœtus, so that the offspring may be in consequence born immune. But no one who thinks clearly would call this a case of inheritance.

(*c*) In many cases it seems possible and useful to draw a distinction between the inheritance of a definite disease and the inheritance of a constitutional predisposition to it. Thus, since tuberculosis is a bacterial disease, since few children are born tuberculous, and since the disease attacks unequally those who are equally exposed to the same external conditions of infection, it seems probable that what is inherited is a blastogenic variation which expresses itself in "vulnerability of the protective epithelia," etc., in short, in a deteriorated power of resistance to the tubercle bacillus. In the same way, to take a case apart from bacterial infection, it seems probable that gout is not as such transmissible, but that what is inherited is a blastogenic variation which expresses itself in an altered mode of eliminating nitrogenous waste,—a constitutional vice which becomes more apparent through excess of food and alcohol.

That diseased states of the nervous system run in families is undeniable, but in many cases they change in particular expression from generation to generation. This points to the position which many hold, which is well argued for by Rohde (1895), that what is really inherited is a germinal variation, which may express itself in general neurasthenia, easy exhaustibility, etc., or under sufficient provocation in some form of acute neurosis. There is no clear case of a normal subject becoming an acute maniac through external shock and transmitting his disease; and Rohde's conclusion, after a careful survey, is that all transmissible nervous diseases have a germinal origin. As Clouston has said: "A neurotic heredity is seen to resolve itself into general morbid tendencies rather than direct proclivities to special diseases." What we have said does not imply that persistent nerve-fatigue and neurasthenia in parents may not favour the outcrop of neurosis in the offspring, for the abnormal nervous condition may through nutritive disturbances affect the germ-plasm (as even Weismann admits), and the fœtus may be readily affected disadvantageously through the mother.

(*d*) The same, or a closely similar diseased state, may arise in different ways, and the heritability will differ with the mode of origin. If the diseased state is inborn in the strict sense, if it be the result of a blastogenic or germinal variation, the probability of transmission is great. But if it has been induced adventitiously by external influences the probability is slight. The distinction is a real one, but it is not always readily drawn in actual practice. Thus the difficulty of distinguishing congenital deafness from that which is adventitious — the result, for instance, of various infectious diseases—may, perhaps, explain why in E. A. Fay's statistics (3078 marriages, 6782 children), the percentage of deaf children in families where both parents were deaf was 8·458; where one parent only was deaf the percentage was larger, namely, 9·856. Where both parents were believed to be congenitally deaf the percentage of deaf children was 25·931; where one parent was deaf congenitally and the other adventitiously, it was 6·538; where both parents were adventitiously deaf, it was only 2·326. Where one parent is congenitally deaf and the other hearing, 11·932 per cent of the children were deaf; where one parent was adventitiously deaf and the other hearing, the percentage was 2·244. In short, it is not evident that adventitious deafness is inherited. Fay's statistics show that deafness among the relatives of the parents increases very largely the likelihood of there being deaf children; and they also seem to show that consanguineous marriages greatly increase the probability of the inheritance of deafness, or of the tendency to it.

(*e*) In many cases there is abundant evidence of the transmission of a specific disease, but this seems always to be what the biologist would call a disease of germinal or blastogenic origin. Thus we may cite a case of hæmorrhagic tendency given by Klebs (quoted by Turner, 1889), where the dark letters stand for affected subjects (all males)·; it illustrates first a diffusion and then a waning of the disease.


M F
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M F F M M M F M M F F M M M F M F M M F M M F
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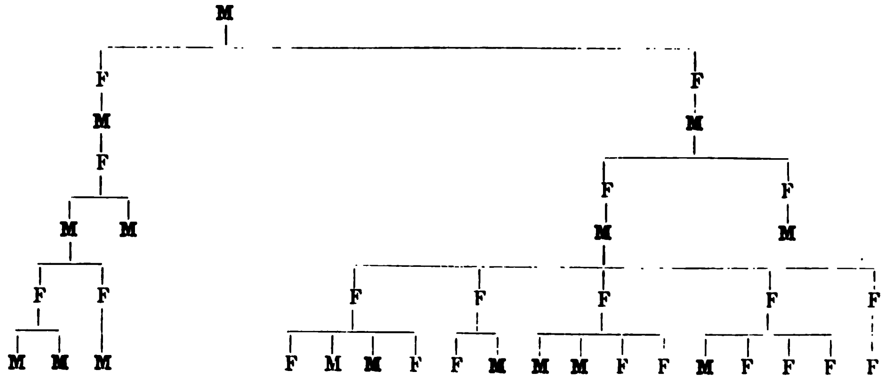
Horner, cited by Klebs, gives a good illustration in reference to Daltonism or colour-blindness.



What has just been noted in regard to specific diseased conditions is likewise true of many abnormalities. Polydactylism has been traced through six generations, and Turner notes a case where a shortening or imperfect growth of the metacarpal bone of the ring finger of the left hand was " traceable throughout six generations, and perhaps even in a seventh, and was, as a rule, transmitted alternately from the males to the females of the family." Scores of examples might be given.

To sum up this section, we may cite four general conclusions from a recent discussion of the subject by D. J. Hamilton :—

(1) There is no evidence proving that diseased conditions of body, excited by external agencies, using the term in its broadest sense, can be transmitted hereditarily through generations.

(2) The various hereditary tendencies or predispositions to disease of the hereditary type have arisen as variations in the germ-plasm.

(3) These predispositions to disease probably extend far back into the history of the human race, and break out only occasionally in accordance with the laws of atavism. [Others would say that the great abundance of presently occurring germinal variations renders it unnecessary to seek for an origin in the distant past.]

(4) External agencies are merely the means of bringing them to light.

When all is said there remains no doubt that morbid conditions and predispositions are inherited, and against this we have to place the probability that relative immunity is also becoming heritable. In the course of natural selection, keenest during the early years of life, the less immune tend to be eliminated, and the standard is thus raised. But in this struggle, as Reibmayr has argued, the most momentous factor is in the external conditions of function and environment, for if these favour the morbid inheritance the organism has to fight a battle with two fronts, which is seldom hopeful. The hope is in the slow increase of constitutional immunity on the one hand and in the securing of wholesome conditions of life on the other. But another side of the problem must not be overlooked, which Haycraft has emphasised in his *Darwinism and Race Progress*—If the race eliminates its own eliminators (the disease germs) which have at least helped to make it what it is, or if it becomes no longer susceptible to their eliminative action, what selective agents—even more discriminating, we may hope—are to take their place?

An article on this subject at the present date ought to close with a reminder that in regard to many problems of heredity and inheritance we are far from being able to formulate general conclusions, and that the hopeful outlook is not in theorising, but in experiment, in the collection of precisely observed data, and in the skilful use of statistical methods.

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Hermaphroditism.

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Hermaphroditism, in the strict and ancient sense of the term—the existence, namely, in the same human being of functionally active testicles and ovaries—has not yet been met with; the name, however, is applied to the cases in which glands anatomically ovaries and glands anatomically testicles have been found in one individual, or, much more commonly, to the cases in which the true sex is masked or rendered dubious by the existence of malformations. There are, therefore, two distinct groups of cases, the first of which contains the anatomically true hermaphrodites (hermaphroditismus verus), while the second includes the individuals of doubtful sex (hermaphroditismus spurius). Of these two groups the latter contains by far the majority of the reported cases which have come to the test of the post-mortem room table and the microscope.

True hermaphroditism may be of three kinds—lateral, bilateral, and unilateral. *Lateral* hermaphroditism may be defined as the presence of an ovary on one side of the body and a testicle on the other, and of this it is claimed that some certain cases have been met with. With regard to that reported by Cramer in 1857, it is stated that there existed a rudimentary uterus and vagina along with, on the right side, a normal ovary, parovarium, and tube; and on the left side a tube, parovarium, and a supposed testicle lying in the scrotal sac. As, however, the microscopical appearances of the last-named body are not given, it is as reasonable to regard it as a herniated ovary. Schmorl's case is better established: it was that of a hypospadiac individual, 22 years of age, in whom, after operation, a small swelling appeared in the left groin, which, when excised, was found to be an ovary; the patient died, and at the autopsy it was discovered that there was a uterus bicornis, and on the right side a testicle with a rudimentary epididymis in the scrotum. Of *bilateral* hermaphroditismus verus, which may be defined as the presence of both an ovary and a testicle on both sides of the body, it must be regarded as doubtful whether a well-established instance has yet been reported. In Heppner's case, a premature, and, in other ways, malformed infant, the external organs were those of the female; while internally there were a rudimentry uterus, a rudimentary vagina, a normal ovary, parovarium, and tube on both sides, and near to each ovary a body containing tubules running towards the hilum, and supposed to be a testicle. *Unilateral* true hermaphroditism must be very rare; in it there are supposed to be a testicle and an ovary on one side of the pelvis, and a testicle or an ovary or neither on the opposite side. Blacker and Lawrence claim to have described the only well-authenticated and genuine case of hermaphroditismus verus unilateralis; but it is to be remembered that it was in a fœtus and not in an adult. The fœtus was well formed save for the genitals; there was a uterus unicornis, a normal tube and ovary on the right side, and on the left side an ovo-testis, with a vas deferens and epididymis. Of course, the whole importance of the case rests upon the demonstration of the so-called ovo-testis; it is claimed by the authors that the gland on the left side was double in nature, that one part of it was ovarian and the other testicular. The microscopical appearances show that in one part there were cell-columns and Graafian follicles with a large amount of stroma, while in the other were tubules filled with cells, forming at the hilum a rete-like structure. Manifestly it becomes very difficult to decide as to the nature of a gland like the testicle or ovary, which in early fœtal life is so similar in structure; arrested development of the testicle might leave a gland not unlike a poorly developed ovary.

Pseudo-hermaphroditism, or hermaphroditismus spurius, is much less rare than the true form; and the explanation is to be found in the embryology of the genitals. Pseudo-hermaphrodites are usually individuals in whom a part of the organogenic scaffolding of the genital organs, which is common to both sexes, persists instead of atrophying; thus in *male* pseudo-hermaphrodites there may be found coexisting the testicles and a uterus and tubes, the Müllerian ducts which ought normally to have atrophied having persisted. But there is never a stage in the embryo in which the scaffolding of both ovaries and testicles exists; hence the rarity, perhaps the impossibility, of true hermaphroditism. The other form of spurious hermaphroditism, pseudo-hermaphroditismus *femininus*, includes the cases in which an individual with ovaries (a woman therefore in reality) has, through adhesion of the labia pudendi, hypertrophy of the clitoris, and a hirsute development on the face, taken on the external appearances of the male. This form, or gynandry, as it is sometimes called, is not so common, and does not carry with it the same social dangers as pseudo-hermaphroditismus masculinus or androgyny; it requires no further description.

Male pseudo-hermaphroditism may be of three kinds. It may be *internal*, when there are testicles in association with external genitals of the male type, and a uterus, vagina, and even tubes. It may be *external*, when there are testicles along with female external genitals and a feminine build of body. Finally, it may be *complete*, when, in addition to testicles, there is a uterus with tubes and external genitals resembling the female type. The commonest form met with is that in which there is scrotal hypospadias (the urethra opening at the base of an imperforate and stunted penis), non-descent or incomplete descent of the testicles, and want of union of the two halves of the scrotum in the middle line. The resemblance to the female type may be intensified by the presence of a vulvar or rather a vestibular canal of no great depth, but simulating the vaginal orifice, and sometimes possessing a hymeneal membrane. Such individuals are usually registered at birth as females, and it is only when puberty arrives that the non-appearance of the menses, and the development of hair on the face and chest, along with other secondary male characters, throw doubt on the real sex of the person, and may lead to a medical examination. In the absence of such an examination it may unfortunately happen that the hypospadiac man is married as a woman, and Neugebauer has reported a number of cases of this occurrence. As in all other malformations, so in this, family prevalence (the occurrence of more than one case in the same family) may be met with, and both Croom and Chiarleoni have given details of supposed sisters who turned out to be hypospadiac brothers. In order to avoid the awkward consequences which may and do follow such erroneous declarations of sex, Lawson Tait has advised that when any doubt as to sex exists at the time of birth the child should be brought up as a boy; if this be done, the risks of error and the dangers resulting from it are much diminished, for male pseudo-hermaphrodites are more common than female, and individuals reared as males are less likely to enter the married state in ignorance of their true sex. Castration has been suggested and carried out in some instances, but it is of doubtful justifiability; redeclaration of sex is attended with many difficulties, as was abundantly shown in Croom's cases; but it is surely preferable in most cases to surgical procedures.

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A HERNIA (ἕρνος = a branch) may be defined as the protrusion of any viscus through an abnormal opening in the walls of the cavity containing it. In general use the word "hernia" is tacitly understood as referring to an abdominal hernia.

In an abdominal hernia the viscus generally derives a complete covering from the peritoneum, and remains always covered by the skin. There are certain regions which from their anatomical confirmation, from congenital incompleteness of development, or from both, are especially predisposed to the occurrence of hernia. These are the inguinal, femoral, and umbilical. Hernial protrusions may also occur in the following situations:—at the obturator foramen, in the perinæum, at the sciatic notch; in the lumbar region and at the linea alba or any portion of the anterior abdominal wall. All such herniæ are known as *external*, in contrast to the *internal*, which occur within the peritoneal cavity, generally into certain pre-existing peritoneal fossæ.

A hernia consists of (*a*) the sac, (*b*) the contents, (*c*) the coverings.

(*a*) *The Sac* consists of peritoneum continuous with and derived from the parietal peritoneum. As a general rule the sac is complete, but in certain cases, for example, "extraperitoneal" cystocele, it may be absent, and in others, such as "paraperitoneal" cystocele, it may be incomplete. Two forms of sac are recognised:—

I. The Congenital, and II. The Acquired.

I. *The Congenital Sac* results from the want of closure of peritoneal processes normal in the fœtus. Such are the *processus vaginalis*, the canal of Nuck, and the peritoneal protrusion at the umbilicus. These will be subsequently referred to in detail. Some forms of sac produced by the traction of an aberrant attachment of the gubernaculum must also be considered as congenital. Such is the sac of an infantile hernia, and such, probably, is the sac formed from one of the pouches of Rokitansky.

II. *The Acquired Sac*, in its earliest stage, is represented by a mere laxity or bulging of the parietal peritoneum. Then at a certain point, the internal abdominal ring, for example, a dimple is formed which, gradually deepening, forms a tube-like process lying in the inguinal canal. The orifice of communication with the peritoneal cavity is now the widest portion of the sac. As the tube-like structure gradually elongates it reaches the upper part of the scrotum, and there, relieved of pressure, expands readily and rapidly, and becomes spherical in shape. To some extent the increase in size of the sac may be the result of the dragging down of peritoneum from the iliac fossa, but it is largely due to the inherent capacity for expansion under a process of stretching possessed by the serous membrane. The upper end lying within the canal is then the narrowest portion of the sac, and is known as the *neck*, the expanded lower end being known as the *fundus*. During the time of the progressive enlargement of the sac adhesions form between it and the structures immediately external, so that when the sac has reached even a moderate size it is fixed, and, except under unusual circumstances, irreducible. The neck of the sac is generally described as being thrown into folds which, from irritative inflammation, adhere to one another, and result in the formation of a constriction, a thick fibrous band, at the abdominal orifice. Such a condition is certainly rare. Since the introduction of the operations of Bassini and Halsted, which impose upon the surgeon the necessity of opening up the inguinal canal and laying bare the neck of the sac, it has been noticed that even if any pleats are present at the abdominal orifice they unfold themselves as soon as the external pressure is relieved. In not a few cases, however, the sub-peritoneal tissue is thicker at the neck than elsewhere, and by a local condensation may form a fibrous ring, but such thickening does not usually affect the peritoneum.

In most cases the interior of the sac is quite smooth, but occasionally adhesions may form, as the result of inflammatory processes, and give rise to irreducibility of the hernia.

If the adhesions affect the mouth of an empty hernial sac and result in its closure there, or if a plug of omentum adhere in the neck of the sac and effectually block the orifice, fluid may accumulate in the interior of the sac so cut off and a *hydrocele of the hernial sac* results.

The sac wall is, in most examples, of the same appearance and thickness as the peritoneum from which it is derived. In old-standing herniæ, especially if an ill-fitting truss or one exerting ill-regulated pressure has been worn, the sac will be found thickened, tough, fibrous, or almost cartilaginous in density.

(*b*) *The Contents.*—Any of the abdominal organs, including the pancreas, may be present in a hernial sac, but in many of the operations undertaken for the radical cure the sac is found empty. The most usual content is probably the ileum, then the omentum, and the jejunum. The large intestine, especially the sigmoid, is not infrequently seen. The omentum generally lies in front of the intestine if both are present. It exhibits a marked disposition to the formation of adhesions to other structures contained within the sac. Adhesions present in a hernial sac may run

1. Between one portion of the sac and another,
2. Between omentum and the sac,
3. Between omentum and intestine,
4. Between intestine and intestine,
5. Between intestine and the sac.

The last are decidedly rare. A thin tag of omentum adherent to the neck of the sac is frequently found on laying open the inguinal canal. Unless the portion of the sac in the inguinal canal be freely opened up, these little tags or threads of adherent omentum are apt to be overlooked. A persisting adhesion of such a kind is not improbably a potent factor in the recurrence of a hernia after operation.

The omentum undergoes certain changes when it has been for any length of time in a hernial sac. It becomes thickened, tough, fibrous and brawny, and in some instances considerably increased in bulk, forming then a hard globular mass connected with the intra-abdominal portion by a more or less narrowed pedicle. Cysts may form, or a calcareous deposit occur, or rarely, malignant disease or tubercle develop. A second spurious sac may be formed by a complete envelopment of the gut in the sac by a thin and stretched omentum. Holes or gaps in this structure have allowed bowel to pass through them, and strangulation has thereby resulted. If intestine alone is present the hernia is known as an *enterocele*, if omentum alone as an *epiplocele*, if both as an *entero-epiplocele.* If only a portion of the wall of the intestine is included in the sac the condition is known as *partial enterocele*, or Richter's Hernia. This is most frequent in femoral hernia. A hernia of Meckel's diverticulum is known as *Littre's Hernia* (not Littré, as frequently written). Other adventitious bodies are occasionally found in hernial sacs, the commonest of these being formed from detached appendices epiploicæ, or tags of omentum which become coated with a thin tunic of fibrous tissue.

(*c*) *The coverings* of a hernia naturally vary with the region in which the rupture exists, but in the same region the conditions presented may vary considerably. Thus in the inguinal region a small hernia, especially a recent one, which has not suffered the pressure of a truss, will reveal on examination all the layers recognised by the anatomist. In an older, larger hernia, where the skin is thickened or chafed by the wearing of a truss, the layers are indurated, fused, and not clearly separable. In the umbilical region, owing perhaps to unrestrained bulging on the part of the contents, the skin becomes thinned, shiny, and almost translucent. In not a few cases ulceration from pressure or friction may extend widely and lay bare the sac and its contents.

CAUSATION OF HERNIA

The causes of hernia are many. There is no single determining factor. The difficulty is not to explain the occurrence of hernia, but rather to understand how any human being goes through a reasonable length of life without suffering from rupture. It is almost impossible to find any subject in the post-mortem room of which it is justifiable to say that in him or her a condition of hernia might not very readily have existed. In all bodies one or more of the "causes" are present. For convenience of description the various conditions leading to hernia may be considered as *predisposing* and *exciting*, or *determining*.

Predisposing Causes.—1. Beyond question the most frequent of these is a congenital aberration of development. Such aberration may affect:—

(*a*) *The Vaginal Process.*—This offshoot from the peritoneal cavity may remain patent or may undergo only partial closure. The "physiological fusion" leading to the obliteration of the process begins earlier and proceeds more rapidly on the left side. Congenital hernia and similar forms of hernia are therefore more commonly noticed on the right side. Probably the partial or complete want of closure of the process and the consequent formation of hernia is far more frequent than is generally conceded.

(*b*) *The Descent of the Testis.*—Any of the forms of retention or ectopy of the testicle strongly predispose to hernia.

(*c*) *The Gubernaculum.*—Irregular attachments of the gubernaculum may pull down into the scrotum accessory sacs, as in infantile hernia. The pouches of Rokitansky seen in the peritoneum, near the internal abdominal ring, may also be formed in this manner. The descent of a congenital cæcocele is induced by traction of the gubernaculum.

(*d*) *The Abdominal Wall.*—Congenital gaps may exist at the umbilicus or at any portion of the linea alba. Congenital weakness of the lower part of the abdominal wall, associated more especially with retention of the testis, is recognised by Macready.

2. *Heredity.*—The children of herniated parents are more likely than others to suffer from hernia. Macready, who has especially investigated this point, concludes that "inheritance is an agent, though perhaps a remote agent, in the production of hernia, and that the influence of the two sexes is nearly equal."

3. *Sex.*—All writers are agreed that males are affected more frequently than females. According to Macready the following are the percentages:—Male inguinal, 83·5; female inguinal, 8·5; female femoral, 5·9; male femoral, 2·1.

Age.—Hernia may be met with at any age. In childhood and early youth congenital causes are at work. During the first year of life an extraordinarily large number of ruptures are seen. Then, year by year, fewer until puberty. From puberty till the termination of the most active period of physical life, at or about fifty years of age, the number steadily increases, but after the latter period gradually declines.

5. *Weakness of the Abdominal Wall.*—In addition to the congenital laxity already referred to, there may also be an acquired feebleness of the muscles. In many cases of inguinal hernia, especially in adults and old people, lateral bulgings of the abdominal wall between the outer edge of the rectus and Poupart's ligament are seen when the patient is standing. There may be also a central bulging of the two recti, and the appearance of *ventre à triple saillée* of Malgaigne is presented. Any form of injury to the anterior abdominal wall may result in the weakening of the damaged area and the protrusion of a viscus through it. A stab, an incision made during an operation, or a blow leading to rupture and subsequent atrophy of the muscle ("Guthrie's Hernia") may all originate a hernial swelling.

The rapid wasting of a large deposit of fat, the wasting associated with old age or with acute illness, may leave the abdominal wall incapable of resisting pressure from within. Parturition, ovarian tumours, ascitic effusion may, by the distension they cause, result in a weakening and a laxity of the whole parieties.

The importance to be attached to the hypogastric bulgings has, I believe, been considerably overestimated by Mr. Lockwood. He remarks: "This bulging always accompanies acquired hernia, and, I believe, precedes its occurrence." Because of this general laxity, Mr. Lockwood further teaches that "curative operations upon acquired hernia are to be avoided." It seems to me that this is an exaggeration both of the frequency of the condition and of the severity of it in relation to operative treatment. Mr. Treves remarks: "In any but aged or broken-down subjects this enfeebled condition of the belly-wall can be greatly improved or even overcome by suitable exercise of the abdominal muscles," a statement which I have verified on several occasions.

6. *The Condition of the Mesentery.*—The normal mesentery is of sufficient length to permit of the entrance of the bowel into an inguinal or a femoral sac. An elongation of the mesentery is consecutive to hernia, and not, as was generally supposed, the initial and responsible defect. Lockwood has shown that "in undoubted cases of acquired hernia, the length of the mesentery is the same as in unruptured people of the same age." As age progresses, the normal attachment of the mesentery to the posterior abdominal wall becomes altered by the gliding downwards of this structure. In its descent the other abdominal viscera accompany the mesentery in what Lockwood terms a compound "prolapse of the mesentery." A profile view of the abdomen of a patient affected with this condition shows a flattening or an excavation above, and a bulging below, the umbilicus. Such a state naturally predisposes very strongly to the onset of a rupture, and if, at the same time, the abdominal wall is lax and enfeebled a hernia is certain to occur. The two conditions are not infrequently associated, and Lockwood believes that both are the expressions of a general tissue deterioration. A rupture, he writes, is "in many cases the local manifestation of a widespread tissue-change."

Exciting or Determining Causes.—The immediate cause of a hernia is any condition which increases the intra-abdominal pressure.

1. *Effort* is the most frequent of these. In the lifting of heavy weights, in straining on coughing or on defæcation, or on micturition when stricture or prostatic enlargement exists, a strain is put upon the abdominal muscles and the cavity enclosed by them is lessened. In children, a tight prepuce, a stone in the bladder, intestinal irritation from bad feeding or from worms and so forth, produce persistent straining. Certain occupations, involving heavy work in certain postures, thus determine the occurrence of hernia. In general it may be said that those trades demanding the most severe exertion show the highest proportion of ruptured persons.

2. Intra-abdominal pressure may be increased by the growth within the cavity of any form of tumour, or by the accumulation of fluid in large quantities. The bowel may, in this manner, be forcibly and powerfully expressed into a hernial sac, even when the patient is bedridden.

THE CONDITIONS OF A HERNIA

A hernia may be reducible, irreducible, incarcerated, inflamed, or strangulated.

REDUCIBLE HERNIA.—*Symptoms.*—A patient, the subject of a reducible hernia of slow formation, will complain (often before any evidence of the hernia is present) of a sense of weight, uneasiness, aching or discomfort in the affected region. This is especially noticeable after exertion, and is relieved by rest. There is, however, a marked difference in individuals in this regard. It is no uncommon thing to see a workman going about his daily task unconcerned by the presence of a gradually enlarging rupture. On the other hand, a bubonocele, trivial, and quite inconspicuous, will give rise to complaints of serious disablement. After a time there may be evidence of intestinal disorder, colicky pains, generally referred to the umbilicus or thereabouts, and dyspeptic trouble. Constipation, especially if the colon is involved in the hernia, may be a prominent, and is occasionally the chief, symptom. Loss of appetite, nausea, and general intestinal discomfort may from time to time be observed, and are the more readily produced by errors of diet, which, under normal conditions, would not be visited with punishment.

Signs.—A reducible hernia, wherever occurring, gives rise to a soft rounded swelling, generally smooth on the surface and regular in outline. On the patient assuming the erect posture, or on straining or coughing, the hernia descends, or, if already down, undergoes such an increase in size as gives rise to an expansile impulse. This "*impulse on coughing*" is present in all hernias, except the strangulated. It is most distinct in enterocele, less so in epiplocele, especially if irreducible, and least so in obstructed hernia.

When intestine is present, the tumour is quite soft and elastic and tympanitic on percussion if of moderate or large size. On applying an even pressure to the tumour it gradually lessens and eventually disappears within the abdomen, undergoing "*reduction.*" If gas and fluid are together present a bubbling sound will be elicited by the pressure and movement; on the reduction of the last inch or two of the bowel there is a feeling as though the gut were being drawn away from one's fingers; these two signs constitute the characteristic "slip and the gurgle" of an enterocele.

When omentum is within the sac, it lies, as a general rule, in front of the bowel. The hernia is then not so smooth, feeling perhaps knotted, lumpy, and irregular. Reduction is slower, and there is neither slip nor gurgle. When the omentum descends again it does so slowly, oozing out of the abdomen evenly and regularly.

Taxis.—For the reduction of a hernia a special manipulation, *taxis*, is employed. The patient is lying upon the back. If the hernia is inguinal or femoral the pelvis may be raised by one or two pillows and the thighs slightly flexed. The neck of the sac in an inguinal hernia, for example, is surrounded by the thumb and fingers of the left hand, and pressure is applied by them downwards towards the fundus to free the neck and to ensure that force applied by the right hand at the fundus does not lose itself in expanding the neck and the upper part of the sac.

IRREDUCIBLE HERNIA.—An irreducible hernia is one in which there is an impediment to the return of the bowel within the abdomen. Irreducibility entails a likelihood of other and more serious complications, inflammation, incarceration, and strangulation.

The causes of irreducibility are:—

(1) *Adhesions within the Sac.*—These in the majority of cases implicate the omentum, and one finds therefore that 90 per cent of irreducible herniæ are epiploceles. Adhesions may involve any of the contents.

(2) *Changes in the Hernial Contents.*—The omentum or mesentery may

increase in bulk from a deposit of fat. Cysts in the omentum or mesentery; tumours of the bowel, simple or malignant; tubercular deposits in the sac or its contents; have all been observed as causes of irreducibility.

(3) *Large size of the Hernia.*—During the course of years a hernia may undergo steady increase, until there is more in bulk outside than inside the abdomen. The abdominal parietes naturally accommodate themselves to the altered conditions, and at the last cannot yield to a degree permitting replacement. The hernial contents suffer from "*perte de droit de domicile,*" as Petit very aptly expressed it. In 10,000 cases Berger found this the cause of irreducibility in 122. In men, inguinal herniæ are affected; in women, umbilical.

The *symptoms and signs* are those of a reducible hernia with the exception of the fixity of the whole or some portion of the hernial protrusion. Irreducibility by keeping open the inguinal canal invites and encourages the descent of further portions of the bowel or omentum. Impulse on coughing is present, though not so clearly as in a reducible hernia. Intestinal worry is often troublesome, and the transverse colon especially may suffer. Symptoms are decidedly more aggressive than in a hernia capable of easy reduction. Irreducibility is not necessarily a permanent attribute.

Treatment.—A hernia whose irreducibility is recent may by appropriate treatment be not infrequently returned within the abdomen.

Confinement to bed, restricted diet, careful regulation of the bowels, and the application of steady even pressure by an elastic bandage with occasional efforts at taxis, have met with fair success. If the patient will not submit to stringent regulations, much good may be done in inguinal hernia by Kingdon's truss. This is in the form of a "hinged cup" and exerts a steady pressure on the herniated mass in a direction opposed to that of its descent. It must be worn night and day, and it possesses the "great advantage that it enables a man to continue at his work whilst his rupture is under process of treatment." In 128 cases recorded by Macready, reduction was effected in 88, sometimes in a few days, sometimes in two or three years. When reduction is accomplished the "hinged cup" is discarded, and an ordinary inguinal or a rat-tailed truss worn. In femoral hernia pressure may be kept up by a truss with a hollowed pad, but reduction in this class of case is neither so frequent nor so speedy as in inguinal hernia.

In cases where reduction cannot be effected some form of truss with an excavated pad or with a "bag" made to fit the hernial swelling, may be worn, in the hope that a further descent of bowel may be thereby prevented. But whatever method be adopted it must be recognised that an irreducible hernia is a constant menace, and may at any moment assume a condition which jeopardises the sufferer's life. Unless, therefore, there are sufficient reasons to the contrary, such as kidney, chest or heart disease, ascites, and so forth, operative measures should be advised if only to render possible and comfortable the wearing of an ordinary truss. The operation for the radical cure of hernia finds its chief justification in cases of irreducible hernia.

INCARCERATED OR OBSTRUCTED HERNIA.—Incarcerated hernia is one in which there is an impediment (*a*) to the return of the bowel within the abdomen; (*b*) to the passage of the intestinal contents along the bowel involved in the protrusion. There is in fact a *local constipation* in an irreducible hernia which becomes turgid and swollen, but retains an impulse on coughing. The condition is generally seen in the bulky herniæ of old people. The bowels refuse to act, there is loss of appetite, the tongue is furred and the breath foul. If unrelieved by enemata and the reasonable employment of taxis, operation must be resorted to or the symptoms of strangulation will slowly develop.

INFLAMED HERNIA—"Hernial Peritonitis."—In this condition there is a localised peritonitis affecting the hernial sac or its contents. The omentum is very generally the seat of this acute condition, which results in many cases from external injury, a blow, unrestrained efforts at taxis, or the pressure of a badly fitting truss. The symptoms and signs are mainly those of local inflammation. The impulse on coughing can be elicited.

General malaise, vomiting, and constipation, if present; are of such slight severity that they can readily be differentiated from the similar but severer symptoms of strangulation. The onset of inflammation is gradual and is attended with fever of varying height. In strangulation the onset is sudden, the development of symptoms is rapid, and there is shock in place of fever.

The patient must be kept in bed, and enemata given until the bowels act well; hot fomentations applied to the hernia will give ease. The diet must for a time be restricted to fluids.

STRANGULATED HERNIA.—A strangulated hernia is one in which there is an impediment (*a*) to the return of the bowel within the abdomen; (*b*) to the passage of the intestinal contents along the bowel involved; (*c*) to the circulation in the imprisoned contents. A hernia is made serious to life only by the possibility of strangulation.

In partial enterocele and in Littre's hernia the obstruction may not yet be complete, either so far as the mechanical conditions or the symptoms are concerned. In a strangulated epiplocele the second condition (*b*) is, of course, not present. Such a condition, however, is rare, and the case would probably be looked upon as an "inflamed" hernia.

Mechanism of Strangulation.—It cannot be said that the precise circumstances under which strangulation occurs in a hernia are properly understood. No question relative to hernia, not even the operative treatment, has had bestowed upon it such a wealth of words as this; and it is in surgery as in finance—much paper and much poverty may co-exist.

An enquiry into the history of most cases (not, of course, cases of partial enterocele, to which the following explanation is not applicable) will reveal an appreciation by the patient of the sudden enlargement of the hernial swelling on exertion. The hernia "comes down bigger than it has ever done before." There is an immediate difficulty in reduction. The hernia is not only more bulky but also tighter, more tense, than it has usually been. In such an increased descent of gut there will also be a larger involvement of mesentery. The mesentery, especially if containing any, even a moderate deposit of fat, is more resistant, more solid, than the elastic and easily compressible gut. In the narrower portion of the neck of the sac (in the inguinal or femoral canals or elsewhere), which may be supposed to be adapted to the quantity of gut or omentum, or both usually traversing it, there will be only the same amount of room for a larger bulk of tissue. The softer and more readily compressible tissues will suffer, and these are the bowel and the veins of the mesentery. There will consequently be venous congestion, and, as Kader's experiments have shown, this will result in a gaseous distension of the bowel involved. Fluid poured out in greater or less quantity as the result of the vascular obstruction will increase the tension in the sac. In this way the condition of strangulation is brought about.

If this explanation were correct we should expect to find the point of strangulation at the most resistant part of the upper end of the sac. In an inguinal hernia the most resistant part is the external abdominal ring; in femoral hernia the crural ring. It is at these two points, in my experience, that the obstruction most usually is found.

In reading the accounts of operations for strangulated hernia we are accustomed to meet with the statement that the obstruction is "at the neck of the sac."

As I have already remarked, a thick fibrous constriction at the neck, though occasionally existing in the old herniæ, is decidedly rare. In an inguinal hernia an operation carried out by any of the methods except Bassini's, Halsted's, or Lockwood's, would not permit an accurate examination of the uppermost part of the sac, and the term "neck of the sac" would probably be held to include anything in the inguinal canal. Now, if in strangulated inguinal hernia the aponeurosis of the external oblique be divided an inch or more above the external abdominal ring, and the fibres separated down to the ring, it can be demonstrated without the possibility of doubt that in the great majority of cases the strangulating factor is the ring itself. Immediately that is divided reduction becomes possible.

That the neck of the sac may sometimes produce strangulation seems clear from the description of specimens of reduction in mass, in which the whole sac has been reduced with the contents still strangled by the cord-like condition at the neck. Such a condition, however, is of extreme rarity. Berger, who investigated the question as to the point of strangulation very fully, so long ago as 1876, wrote that the neck of the sac was "rarely capable of forming a veritable stricture."

In femoral hernia the crural ring is the obstructing agent; division of Gimbernat's ligament generally permits of reduction. In umbilical hernia the obstruction is formed by the tight fibrous ring outside the neck of the sac.

In some, comparatively rare, examples, there may be an acute strangulation *within* the sac as the result of the passage of a loop of bowel through a hole in the omentum, as the result of a volvulus at the neck of the sac, or as the result of the nipping of the gut by an adhesion within the sac. Under these circumstances there is an acute obstruction in a hernial sac unconnected with the hernia as such.

Pathological Changes in the Hernial Contents as the Result of Strangulation.—When a hernia becomes strangulated, the venous channels become engorged. Blood can still enter by the thicker and less compressible arteries when it can no longer return through the veins. The congestion leads to an exudation of fluid, the colour and general quality of which depend upon the severity of the pathological changes in the bowel. The gut involved becomes blue and livid in appearance, and its colour deepens by degrees until it becomes rich purple, and finally black or ashen grey. Small extravasations appear as the result of the rupture of distended venules; in the mesentery the extravasated blood may form a solid slab of clot. The natural lustre of the bowel is retained for some time, but as the wall becomes thickened and œdematous, the serous membrane looks dull and turbid, and flaky masses of lymph adhere to its surface. Gangrene may affect the whole of the imprisoned loop, or just that ring of it subject to the keenest pressure at, or near, the neck. This point especially should receive careful examination after division of the structure, and for that purpose the bowel above and below the snared loop must be pulled down. The indentation made by the stricture is then readily seen, and can if necessary be dealt with.

When the epiploon is involved, congestion may pass on to gangrene, but does so very rarely. When omentum is present in a hernial sac, adhesions almost always form, and by them blood-vessels may carry an alternative supply of blood.

The fluid in the sac is at first thin and serous, but becomes by degrees more and more deeply tinged with blood; turbidity is soon noticed, and as soon as the bowel wall is damaged there is an escape of the bacterium coli commune. In the fluid this organism flourishes, produces harmful toxic products, and gives rise to a peculiarly foul and penetrating odour. The character of the fluid is a good index to the amount of damage the intestinal wall has suffered.

In amount the fluid varies considerably. When bowel is in the sac, there is always a fair quantity, but when omentum alone is present, or when a solid viscus such as the ovary is present, the fluid is less. Macready asserts that in 33 per cent of cases of strangulation no fluid is present, an estimate which I consider to be greatly in excess of the truth.

As the fluid becomes more putrid it affects not only the sac, but the coverings of the hernia, which become red, inflamed, and œdematous, and may in rare cases go on to gangrene and the formation of a fæcal fistula, the clumsy and unpleasant result of "Nature's cure."

Bacteriological examinations of the fluid have been carried out by many observers. Clado has found the bacterium coli commune to be most frequently present, and in cases of death from strangulation has observed the organism in the spleen, liver and kidneys, and in larger numbers and more frequently in the lungs. Barbacci has recognised Fränkel's diplococcus, and the staphylococci pyogenes albus et aureus and streptococci have also been found. Brentano asserts that after strangulation has existed more than twenty-four hours, micro-organisms are constant. Weichselbaum considers the diplococcus pneumoniæ as the occasional cause of acute general peritonitis.

Changes in the Bowel above and below the Hernia.—The bowel above the hernia becomes distended to a degree dependent in part upon the duration and severity of the obstruction. Certain changes occur in it, which have been carefully described by Kocher. The bowel becomes blue in colour from venous stasis, ecchymoses may form in the mucous membrane or beneath the serous coat, nutrition is altered and the protective influence of the epithelium is destroyed. Absorption of bacteria and of the toxic products of the intestinal contents begin from that moment, and the patient, unless relieved, may die either from general intoxication with symptoms of weakness of the heart and collapse, or from local intoxication with necrosis of the mucous membrane, beginning in the ecchymosed patches and going on speedily to ulceration and perforation.

The Symptoms of Strangulated Hernia.—The symptoms of strangulated hernia are very similar to those of acute intestinal obstruction. The first occurrence noticed is the forcible descent of a larger mass of hernia than is usual, or the decided increase of a rupture that is irreducible. This *accession of bulk* is attended with *pain* in the hernial swelling, chiefly at the upper part and in the abdomen near the umbilicus, at first and for a brief period intermittent, but soon becoming and remaining continuous. The pain is very acute, with exacerbations of a colicky nature; it causes the patient to be bent double, to sweat profusely, to become collapsed and faint, and it rapidly induces nausea and vomiting.

Vomiting is the chief and most important symptom. It is unvarying, distressing, and exhausting. At first the stomach is emptied, then the duodenum, the vomit being bile-stained; finally, the decomposing and fœtid contents of the distended gut above the obstruction are ejected. So pungent and putrid is this vomit at the last, that it is described as "fæculent" or "stercoraceous," implying that fæces are contained in the vomit. This is rarely, if ever, the case, the odour and appearance being in fact caused by the bacterial profusion in the over-distended bowel. Hiccough is sometimes present, and is an untoward symptom. The temperature as a rule is subnormal; the pulse is rapid, thin, and wiry. Constipation is absolute and persistent: enemata may empty the bowel below the obstruction, and a little "flatus" accidentally introduced by the syringe may be passed, but neither fluid nor solid leaves the hernial sac. Even in partial enterocele the same completeness of obstruction is observed in the majority of the cases, as the result it may be of reflex paralysis of the gut, or of kinking at the hernial orifice. In others there may be the passage of flatus and occasionally of the liquid contents of the gut. The abdomen is generally a little prominent, is very rigid and tender on pressure. No distended or contracting intestinal coils can be observed. The *appearance* of a patient suffering from strangulation is generally characteristic. There is at first an unhealthy almost livid flushing of the face, soon giving place to pallor with shrinking and "drawing" of the features, and a fixed and meaningless staring of the eyes. The face and limbs are covered with a sweat, and feel cold and clammy to the touch. There is a tinge of blueness in all the skin. The expression of the face becomes anxious, and speaks of terror and impending disaster. The body looks, and is, desiccated, shrivelled, parched for lack of any water. The tongue is dry, harsh, and brown; thirst is intolerable. The urine is diminished in quantity, and indican may be present. In cases of intense strangulation of the small intestine, albumen may also be found. According to Englisch it is present in two-thirds of the cases operated upon, and is a sign of some importance. The symptoms of toxic poisoning are seen in their most striking form, and in a state of gradually deepening collapse the patient dies.

The Signs of Strangulated Hernia.—The hernial swelling is larger, often considerably larger, than is usual. It is tense and hard, and irreducible. There is no impulse on coughing. Manipulation of the tumour is resented, owing to extreme tenderness. If strangulation persists, the surface becomes of a livid or dusky red colour, which gradually deepens until a black slough is formed. Emphysema may be felt, and perforation of the gut and of the skin may in the end lead to the formation of a fæcal fistula.

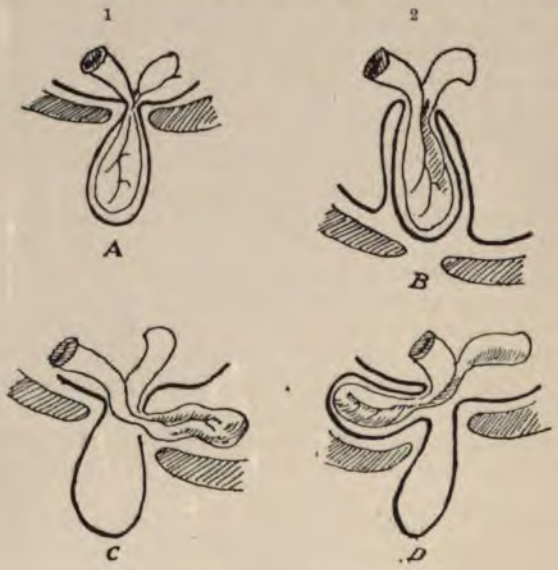
Treatment of Strangulated Hernia.—The hernial contents must be returned within the abdomen at the earliest possible moment. Reduction may be effected by means of taxis aided by the hot bath or an anæsthetic, or by operation. Taxis, unless employed with discrimination and for a brief period, is probably more harmful than operation.

False Reduction.—Taxis, if employed without constraint, or occasionally even when judiciously applied, is attended with some risks, such as injury, bruising, or laceration of the sac or its contents, or the forcing of infected fluid into the peritoneum. The most serious accident, however, is that known as *False Reduction.* I have elsewhere discussed this subject fully; it is not necessary here to do more than epitomise my conclusions. False reduction may be described as of the following varieties:—1. Reduction into a sac lying between the peritoneum and the transversalis fascia. This sac has probably received the bowel on many previous occasions. It exists in both the inguinal and femoral regions, forming the special class of hernia known as properitoneal or bilocular or the "displaced" hernia of Bryant. This sac may pass (*a*) inwards to the bladder, (*b*) backwards to the iliac fossa, or (*c*) outwards towards the anterior superior spine. This form is probably by far the most frequent. In a very few cases, not five in all, the apparent reduction has occurred into one of the interstitial sacs beneath or superficial to the external oblique. Such a case was that under the care of Mr. Cock (Guy's Hospital Museum, No. 1120). 2. Reduction of the whole sac within the abdomen but outside the peritoneum, the relations of sac and contents to one another being unaltered and strangulation at the neck still persisting. It is probable that the loosening of the sac is of long duration and is the result of vigorous and ill-regulated taxis. This variety is rare. It was first described by Saviard, afterwards by Le Dran, Lafaye, and Richter. 3. Rupture of the neck of the sac and escape of the contents of the sac through the rupture into a cellular space outside of the peritoneum. The rupture generally occurs at the back of the sac just below the neck. This form of reduction has been well described by Birkett. If fluid alone escapes while the bowel remains, an illusory appearance of reduction will be presented. If in incomplete "false" reduction the rupture is at the distal extremity of the sac and the discharge of fluid subcutaneous, it is impossible, of course, for bowel to escape. As a variety of this form may be mentioned the passage of the strangled intestine through a cut made near the neck of the sac when a hernia knife had been introduced to divide the "stricture at the neck of the sac." Maydl has observed this accident. After the supposed division of the strangulating band, pressure is applied to the bowel, and reduction apparently occurs. The neck of the sac is, however, untouched, and the strangulation consequently persists. A very beautiful example of this kind is recorded by Farabœuf. This form also is rare.

A fourth form of "reduction in mass" is described and figured by Roser and mentioned by Pick. It consists in the reduction "into the inguinal canal" of the bowel that has formerly occupied the scrotum. A wide dilatation of the canal is considered as permitting this partial reposition. I have not been able to discover a museum specimen of such a condition, nor have I found any unequivocal case in the literature of this subject, though mention of the condition is made by several of the earlier writers on hernia. Chelius considered that in herniæ of moderate or large size, when reduction was apparently complete, a small segment of gut, thick with inflammatory products and paretic, might lie inert in the canal and undergo a later, secondary strangulation. Reduction of the strangulated contents of a hernial sac into a second underlying sac in the scrotum has been recorded by Armand, Horn, Sandyfort, and Scarpa. It is possible that these second sacs are pulled down into the scrotum by gubernacular bands, in the same manner as the sac of an infantile hernia. Demmeaux has suggested that whether the sac first present in the scrotum be congenital or acquired, the press of intestine on a weak and loosely supported neck may cause a diverticulum slowly to develop. Such pouch may be in front or behind, to the inner or outer side. If rupture of the neck of a femoral sac occurs the bowel may pass into the subperitoneal tissue. Callisen recorded in 1777 a case where, owing to rupture of the sac below the neck the bowel had escaped and made for itself a lodgment between the pectineus and psoas muscles. These conditions may be represented in tabular form thus:—

Complete False Reduction.—1. Reduction into a properitoneal sac; rarely an interstitial sac. 2. Reduction of the whole sac with contents within the abdomen, outside the peritoneum, the mutual relations of sac and contents being undisturbed. 3. Rupture of the neck of the sac or its division during operation and extravasation of the hernial contents into the subperitoneal tissue.

Incomplete False Reduction.—1. Rupture of the sac at the neck or elsewhere with escape of fluid only. The hernial swelling becomes considerably smaller, but the bowel remains strangled. 2. Reduction of part of the hernial contents. A small portion remaining in the canal, paralysed and stiff with inflammatory products, undergoes a "secondary" strangulation.



Complete False Reduction.

Fig. 1.—A. Sac and contents in normal position.
Fig. 2.—B. Sac and contents displaced "en masse."
Fig. 3.—C. Rupture of sac near the neck.
Fig. 4.—D. Properitoneal sac.

If taxis prove ineffectual, operation must be resorted to. It is not possible to exaggerate the evils of delay, in cases of strangulation. There is nothing to be gained by delay, there is everything to be lost. Operation in itself is devoid of serious risk. Including all cases, the aged, infirm, or bronchitic, death would probably not occur in more than 5 per cent if operation were adopted at the earliest moment in cases of inguinal and femoral hernia. The mortality after strangulated hernia is considerably greater than that. During the last fourteen years at the Leeds infirmary, since a statistical report has been published, the mortality has been, for strangulated inguinal hernia, 17·4 per cent, and for femoral hernia 23·8 per cent. The difference between these and 5 per cent is the *mortality of delay.* During the same period the operation for the radical cure has had a mortality in inguinal hernia of 2·3 per cent, in femoral hernia of 1 per cent. If the last five years only are selected the mortality is in both cases less than 1 per cent.

Treves gives the mortality of strangulated hernia as "over 30 per cent," Ross and Carless at "about 35 per cent." Now taxis, even if successful, so far as the reduction of the hernia is concerned, is not without its dangers. Bryant estimates the mortality after reduction by taxis in inguinal hernia 3·8 per cent, and in femoral hernia 10·5 per cent. It would probably be in accord with the experience of most hospital surgeons to say that taxis applied for one or at the most two minutes is sufficient to reduce a hernia that can be with safety reduced. A longer application than this will produce such damage in the gut as to render its reposition unsafe. When the patient is fully prepared for operation, and under the anæsthetic, a further brief attempt at reduction may be made, but failing then, the surgeon should proceed at once to operation.

Umbilical hernia, whether strangled or not, is a much more serious matter. The operation for the radical cure with us during the last fourteen years has had a mortality of 6·5 per cent, for strangulated hernia the mortality has been 56·4 per cent. The argument for early operation is therefore equally valid here.

Details of the Operation.—Immediately the hernial sac is opened the fluid contained therein should be emptied out and the sac with its contents washed gently but efficiently with hot sterile salt solution. The gut is then inspected.

A. If found to be living, the strangulating agent should be divided and the loop of bowel pulled further out of the abdomen, in order that the line of constriction may be examined. On no account should the finger be introduced into the neck of the sac with the idea of stretching the ring to permit more readily of replacement. If the deep groove which marks the point of constriction be not seriously damaged the bowel may, with gentle manipulation, be returned. If there is a line of ashen-grey slough this may be infolded by a layer of Lembert's or Halsted's sutures, and the gut replaced. Any omentum present may then be dealt with. Adhesions are often present, but it is not necessary to separate them, a process which is both slow and tedious. Traction on the omentum will bring the non-adherent part above the constriction into the sac; a series of ligatures is applied, and the omentum cut across below them. The neck of the sac is then ligatured and divided, and the sac and the adherent distal portion of the omentum are together stripped up and removed. A radical cure may be performed then by any of the usual methods.

B. If found to be in a doubtful condition the loop may be drawn, after division of the constriction, well down into the wound, and left there a few minutes for inspection. If there is any evidence of a gradual return of the natural colour the gut is still living; if there is none, and especially if the veins of the mesentery are felt to be blocked, the gut, if not dead, is certainly doomed. If, however, doubt still lingers, the gut may be returned just within the abdomen (where, owing to its paresis and the rapid formation of filmy adhesions, it will remain), and a tube passed down to it. Experience has abundantly shown that if the gut should give way its contents will in general pass along the hernial track and a fæcal fistula result. Or, and preferably, the loop of bowel may be drawn outside the wound, covered with a protective dressing, and left for 24 or 36 hours, then re-examined and dealt with according to its condition.

C. If found to be gangrenous two courses may be adopted.[1]

1. The strangled loop, with the implicated mesentery, may be excised, and the divided ends of the bowel stitched up, with or without a mechanical appliance.

2. The bowel may be opened with or without division of the constriction and an artificial anus formed. As modifications of this procedure may be mentioned the introduction of a Paul's tube after slitting open the bowel, or the stitching of the opened gut to the skin. In not a few cases where the gut has been laid open without division of the stricture the acute obstruction has continued. A careful division of the stricture without undue disturbance at the upper part of the sac can do no harm, and is therefore generally desirable.

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[1] The method of Helferich has been so seldom practised that its value is not yet determined. It consists in drawing out a loop of gut and uniting the ends by means of a Murphy button. The loop is then left, covered by an antiseptic dressing, and resection performed when the patient has rallied.

Which of these two courses is to be adopted must depend upon many conditions to be carefully weighed by the surgeon. It must be admitted at once that *whenever possible resection should be performed.* A notable argument has been made by many of the more ardent surgeons in a reference to statistics, showing that the mortality after resection is less than after the formation of an artificial anus. Thus in 394 cases of gangrene that I have tabulated, where an artificial anus was made, 30·7 per cent of patients recovered. In 443 cases of primary resection 53·9 per cent recovered, a difference of more than 20 per cent in favour of resection. Nothing could be more misleading than this argument. Resection is done in the less severe cases by surgeons adept and skilful in manipulation, with the most desirable surroundings and with adequate help. A surgeon who resected intestine in a moribund patient could have no sense of the fitness of things. In such a case the laying open of the gut would be performed to give the patient a last, slender chance; if death followed it would be due to the delay, not to the method. I hold the argument from statistics, therefore, as not valid, though it supports the method which is ideally the better. There are certain circumstances under which enterectomy, with stitching of the divided ends, could not be legitimately performed. These are—

(α) When the patient is profoundly collapsed from septic poisoning, almost pulseless and moribund; or aged, bronchitic, or otherwise in bad condition.

(β) Where prolongation of the operation would add a serious risk, as in cases where a large segment of gut is involved (as much as 6 feet has, however, been successfully removed).

(γ) Where strangulation has been of long duration. After 3-4 days' obstruction resection is rarely successful. In such cases the distension of the gut above the constriction is considerable, and the bowel does not lend itself readily to successful suture.

When, however, one meets with a case of recent, limited gangrene in a young, otherwise healthy subject, not exhausted by vomiting nor collapsed by the absorption of poisonous material, resection will find its most successful application.

The disadvantages of the method in which an artificial anus are formed are neither few nor inconsiderable. Briefly they are:—

(α) The possibility of the continuance of symptoms of acute obstruction, a possibility by no means remote, if the stricture is not adequately divided.

(β) Septic infection from the wound in the gut or from the sac spreading to the peritoneum, or infecting a wide area surrounding the opening.

(γ) The opening in the gut may be high up in the small intestine, and inanition may ensue.

(δ) The necessity of a second operation, which is not without its own mortality.

We are not, however, in a position to say that the application of either method should be unvarying. Each has its uses. No hard and fast rule can be made; the surgeon can only be guided by the general principles laid down; the decision he makes will depend upon his own resourcefulness, and his readiness and capacity to appreciate the relative value of many signs.

Complications of Strangulated Hernia.—Among the more important of these may be mentioned—

1. *The persistence of symptoms after reduction,* due to—

(*a*) False Reduction, complete or incomplete (see page 502).

(*b*) Acute enteritis in the snared loop leading to paralysis, and going on perhaps to ulceration, gangrene, or perforation.

(*c*) Acute peritonitis from infection by the damaged gut or by the escape of infected fluid from the sac.

(*d*) The existence of acute strangulation, apart from the hernial protrusion, or connected with it.

(*e*) The existence of another hernia, femoral, obturator, etc., which is strangled.

(*f*) The existence of a condition where symptoms are mistaken for strangulation in a hernia, *e.g.*, lead poisoning.

If (*b*) or (*c*) are present the abdomen should be opened; if the former condition is found an artificial anus should be made; if the latter, the cavity may be flushed and drained.

2. *Affections of the Lungs.*—Pneumonia and bronchitis are seen, not infrequently, in cases of strangulated hernia both before and after reduction. Verneuil, in 1881, called attention to an intense pulmonary hyperæmia as a factor in causing death. Gussenbauer regarded the condition of the lungs as the result of infective embolism from the mesenteric vessels; Lesshaft suggested that infection was probably conveyed by inhaled particles of putrid vomit, especially during anæsthesia.

The signs of pulmonary trouble do not, as a rule, become manifest till 12, 24, or 36 hours after reduction. There is at the first some respiratory concern, hardly amounting to difficulty, a little hurry in the rate of breathing, a trifling elevation of temperature. In a few hours all these conditions are accentuated, and pneumonia, usually of a very aggressive type, is developed. The investigations into the bacteriology of strangulated hernia are of interest in this connection (see page 501).

As more or less remote sequelæ of strangulated hernia should be mentioned *stricture of the intestine in the strangled loop, and acute intestinal obstruction from the formation of peritoneal adhesions.*

INGUINAL HERNIA

Inguinal Hernia is by far the most common form of hernia met with. According to Macready, in 100 men ruptured, 97·5 per cent have inguinal and 2·5 per cent femoral hernia. Among 100 women ruptured, 60·3 per cent have inguinal and 39·7 per cent femoral hernia. Among 100 ruptured persons of both sexes the following proportions obtain:—Male inguinal, 83·5 per cent; female inguinal, 8·5 per cent; female femoral, 5·9 per cent; male femoral, 2·1 per cent.

Anatomy of Inguinal Hernia.—An inguinal hernia leaves the abdomen by the inguinal canal, entering at the internal abdominal ring and emerging at the external. The canal is 1½ in. long in the adult. The anterior wall is formed by the aponeurosis of the external oblique, and in the outer third by the fibres of the internal oblique arising from Poupart's ligament.

The posterior wall is generally described as being formed in its inner two-thirds only by the conjoined tendon, but this statement needs correction. The conjoined tendon forms the inner part of the posterior wall, but in the outer part there is, in the great majority of cases, a quite distinct layer of tendinous fibres derived from the transversalis muscle. This layer (sometimes referred to as the "reflected tendon" of Cooper) extends up to, and forms, the inner boundary of the internal abdominal ring, where there is a noticeable thickening, the "ligament of Hesselbach" descending from the outer limb of the fold of Douglas.

The posterior wall is also formed by the triangular fascia of Colles, sometimes referred to as the "posterior" pillar of the external abdominal ring, and the transversalis fascia, here thickened by the addition of bundles of oblique and transverse fibres. The conjoined tendon is a very variable structure. In the great majority of cases it is thin and weak, and blends internally with the sheath of the rectus.

The floor of the canal is formed by Poupart's and Gimbernat's ligaments, and the roof by the arching fibres of the internal oblique and transversalis.

Action of the Inguinal Canal.—The term "canal" is perhaps unfortunate if it conveys the idea of an open tube transmitting the cord, for nothing of the kind exists when the conditions are normal. In a healthy individual the anterior and posterior walls are in close apposition. When the arched fibres of the internal oblique contract, they become straight and descend towards Poupart's ligament, compressing the spermatic cord against that structure. While so descended they are gripped and fixed by the pressure of the anterior and posterior walls, which, by the contraction of the external oblique and the transversalis, are made tense and are approximated. The canal therefore acts as a "sphincter," and would, with greater accuracy, be described as the *Inguinal valve.* For the efficient working of this valve it is necessary that the "canal" should be empty. Any structure, such as a deposit of fat along the cord, or a pleated sac after operation, which occupies the canal, will prevent the descent of the arched fibres of the internal oblique and transversalis, and thereby make impossible the normal sphincteric or valvular action of the canal.

If the lower portion of the anterior abdominal wall is examined from behind certain elevations with intervening depressions will be noticed. Along the middle line a fold, the *plica umbilicalis media,* extends from the bladder along the urachus to the umbilicus; on each side of this there is a ridge, the *plica umbilicalis lateralis,* raised up by the obliterated hypogastric artery, and still further outwards a slighter fold, the *plica epigastrica,* indicating the line of the deep epigastric artery. Between the two first folds is a recess, the *fossa supra vesicalis,* between the second and third the *fovea inguinalis mesialis,* and to the outer side of the plica epigastrica the *fovea inguinalis lateralis.* Occasionally the line of the obliterated hypogastric coincides with that of the deep epigastric, and only a single ridge is raised up.

The plica epigastrica, as it passes upwards and inwards, forms, with the outer edge of the rectus to the inner side and Poupart's ligament below, a triangular interval known as the *Triangle of Hesselbach.*

An oblique inguinal hernia descends, outside the plica epigastrica, through the fovea inguinalis lateralis. A direct inguinal hernia may pass (*a*) between the plica epigastrica and the plica umbilicalis lateralis, or (*b*) between the latter and the central fold; the former is known as a *superior* or *external,* and the latter as an *inferior* or *internal* direct inguinal hernia.

The coverings of an oblique inguinal hernia are skin, superficial fascia, intercolumnar fascia, cremasteric fascia, infundibuliform fascia, subperitoneal tissue, and peritoneum. Of a superior or external direct hernia, skin, subcutaneous tissue, intercolumnar fascia, cremasteric fascia, and some fibres derived from the tendon of the transversalis muscle at or near the ligament of Hesselbach, transversalis fascia, subperitoneal tissue, and peritoneum. Of an inferior or internal direct hernia, skin, subcutaneous tissue, intercolumnar fascia, Colles' triangular fascia, conjoined or transversalis tendon, transversalis fascia, subperitoneal tissue, and peritoneum.

The following varieties of inguinal hernia will be described:—

Oblique Inguinal	Superior Direct	Inferior Direct
Skin	Skin	Skin
Superficial fascia	Superficial fascia	Superficial fascia
Intercolumnar fascia	Intercolumnar fascia	Intercolumnar fascia
Cremasteric fascia	Cremasteric fascia	Colles' fascia
Infundibuliform fascia	Transversalis tendon	Conjoined or Transversalis tendon
Subperitoneal tissue	Transversalis fascia	Transversalis fascia
Peritoneum	Subperitoneal tissue	Subperitoneal tissue
	Peritoneum	Peritoneum

(A) OBLIQUE INGUINAL HERNIA.

I. The acquired form.

II. Hernia depending upon congenital anomalies in the processus vaginalis.

(*a*) Congenital Hernia; (*b*) Hernia into the funicular process; (*c*) Infantile Hernia; (*d*) Encysted Hernia; (*e*) Hernia with retention of the testis; (*f*) Hernia into the canal of Nuck.

III. Properitoneal Hernia.

IV. Interstitial Hernia.

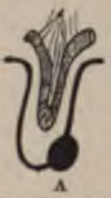
(B) DIRECT INGUINAL HERNIA.

A. OBLIQUE INGUINAL HERNIA

I. *Acquired Inguinal Hernia.*—This form of hernia generally makes its first appearance in adult life. When confined to the inguinal canal the swelling is known as a bubonocele; after escape from the canal it becomes a *scrotal* or *complete* hernia. The sac is formed slowly and is derived from the parietal peritoneum. When the hernia reaches the external abdominal ring, it is free to enlarge in all directions. The result is that a globular scrotal swelling is connected to the abdomen by a narrow neck; the neck being the portion lying in the canal. As the tumour enlarges, the neck (as a result of the approximation of the internal to the external abdominal ring from traction exerted by the hernial mass) becomes progressively shorter. The scrotal portion descends down to, and enlarges in front of, the testis. The sac is never intimately adherent to the structures of the cord.

II. *Hernia depending upon congenital anomalies in the processus vaginalis.*—These forms are probably much more common than is generally believed. There may be little or nothing to distinguish an ordinary (so-called acquired) hernia from a hernia into the funicular process, and Russell has recently suggested that all herniæ are congenital in the sense that they occur in preformed sacs derived from the processus vaginalis. It is certainly quite common to find in adult bodies a fine funicular process unclosed when there has never been a history of any hernial descent. If the upper portion of this process remained open and formed a hernial sac, the case would almost certainly be looked upon as acquired, though in reality dependent upon congenital abnormality in the processus vaginalis.

(*a*) *Congenital Hernia* is characterised by the absence in the processus vaginalis of any attempt at closure. The process remains open throughout and forms a ready-made hernial sac into which the gut descends without hindrance. In the sac the hernial content is in touch with the testis. A hernia of this form frequently appears quite suddenly, and in a young adult is not uncommonly strangulated at its first emergence. Constrictions, the result of attempted closure at certain points, may give rise to an "hour-glass" sac. The most common narrowing of this kind is situate at the upper end of the tunica vaginalis.



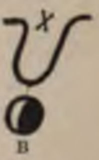


FIG. 5.—A. Congenital hernia.
FIG. 6.—B. Hernia into the funicular process.

(*b*) *Hernia into the funicular process* results when the tunica vaginalis is fashioned from the lower part of the processus, while the upper part remains unchanged. This hernia possibly forms the majority of the cases of inguinal hernia.

(*c*) *Infantile Hernia.*—The term "infantile" has no reference to the period at which the hernial tumour first appears. The admirable work of Lockwood in this matter has been generally accepted by all subsequent writers. This author first described four varieties of infantile hernia. In each of them a second peritoneal process is pulled down into the scrotum behind the processus vaginalis, by an aberrant attachment of the gubernaculum. The varieties of infantile hernia depend not upon the condition of this second peritoneal tube, but upon the varying states of closure in the processus vaginalis, according as to whether this process is (1) closed below and open above; (2) closed above and open below; (3) closed above and below; (4) open throughout. I have seen one example of an infantile sac, closed at its upper end, containing hydrocele fluid. In the unclosed processus vaginalis a congenital hernia had formed.

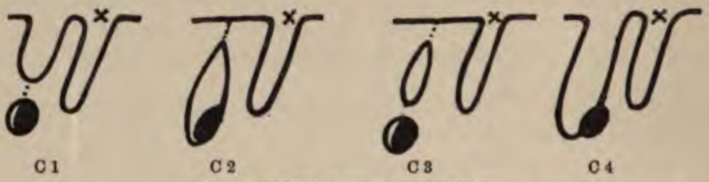


FIG. 7.—Infantile hernia,—4 varieties.

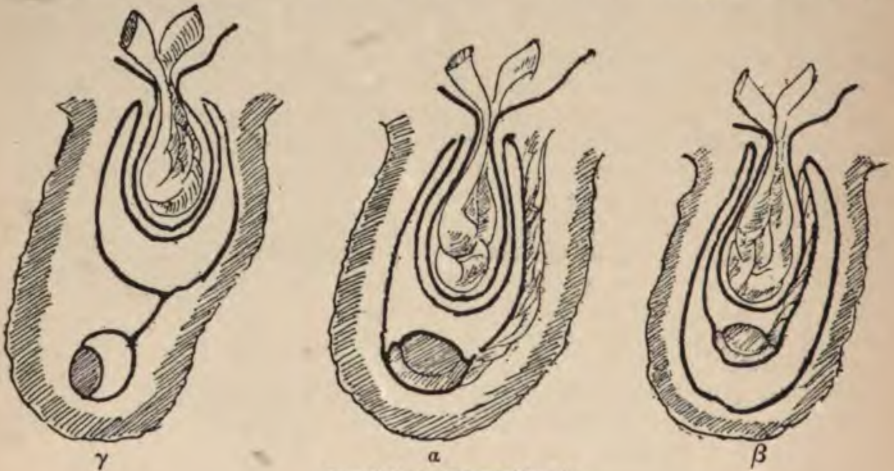
(*d*) *Encysted Hernia.*—I described in my "Arris and Gale" Lectures three forms of encysted hernia—

α. Encysted hernia with the testis lying at the bottom of the sac.

β. Encysted hernia with the testis lying at the apex of the hernia.

γ. Encysted hernia of the funicular process.

α. This form depends upon the closure of the vaginal process only at the internal abdominal ring. A hernia now develops, the sac being formed from the parietal peritoneum, and as it descends reaches the upper end of the large tunica vaginalis. This it dimples and then, in its steady increase, invaginates until the hernial sac hangs pendent from the internal abdominal ring, in the cavity of the tunica vaginalis.



FIGS. 8, 9, 10.—Encysted hernia.

β. This form is similar to α, except that it is associated with retention of the testis in the inguinal canal. The hernial sac in its descent meets with the testis, which it carries before it and invaginates into the tunica vaginalis.

γ. This form is the same as α, except that the tunica vaginalis is shut off and the hernia is encysted in the funicular process.

(*e*) *Hernia with retention of the testis* is not unusual. The testis as a rule is retained in the inguinal canal or at the external abdominal ring. The inguinal valve is prevented from acting, and the descent of a hernia is thereby invited. The testis is generally small and soft, almost always atrophied, and is at times exceedingly sensitive. Cases of hernia with retention of the testis may be treated by the application of a truss, the testis being disregarded, but are more satisfactorily dealt with by castration and the performance of a radical cure.

(*f*) *Hernia into the canal of Nuck.*—If the process of peritoneum accompanying the round ligament in the female remain unclosed, it forms the "canal of Nuck." A hernia into this process corresponds to a congenital hernia in the male.

III. A *Properitoneal Hernia* fulfils the following conditions—

i. The hernial sac has two loculi.

ii. The inner loculus lies between the peritoneum and the fascia transversalis.

iii. The outer loculus lies in the inguinal (or crural) canal, or in rare cases between the layers of the abdominal wall.

iv. Both loculi open into the abdomen by a single orifice, the "ostium abdominale."

The inner loculus may occupy one of three positions—

1. It may pass upwards and outwards towards the anterior superior spine.
2. It may pass directly backwards and occupy the inner part of the iliac fossa.
3. It may pass downwards and inwards into the true pelvis and lie by the side of the bladder.

The right side is more commonly affected than the left. So far as causation is concerned little is accurately known. The evidence may be thus summed up. The hernia arises most frequently as the result of some congenital abnormality in the inguinal region—as an added factor there may be a loosening (congenital in origin, Linhart) of the parietal peritoneum. In some cases a more or less complete "reduction in mass" may be the responsible factor. In a few cases the inner sac may be first formed, and the inguinal sac be a secondary diverticulum. (For further details see the report of my lectures, *Lancet*, Feb. 24, 1900.) The peritoneal conditions are similar to those found in "bilocular hydrocele."

IV. An *Interstitial Hernia* fulfils the following conditions—

1. The hernial sac has two loculi.
2. The inner, upper loculus lies (*a*) between the internal and external oblique (interparietal), or (*b*) between the external oblique and the skin (extra-parietal).
3. The outer, lower loculus lies in the inguinal canal.
4. The upper loculus opens into the lower in some cases close to, in other cases a little distance from the internal abdominal ring.

In 73·4 per cent of cases in males there is some abnormality of the testis. The hernia is, however, of decidedly greater relative frequency in females than in males. Macready gives ·13 per cent of all cases in males and ·61 per cent in females.

The development of the interstitial sac in males depends almost certainly upon the incomplete testicular descent. The testis bars the way to a further protrusion of the hernia, and the inguinal sac therefore bulges out in the direction of least resistance. This explanation is strengthened by the observation, which is almost universal, that the scrotum is hardly ever fully occupied by a sac containing bowel or omentum. In 129 cases observed at the Truss Society, in only 9 was the scrotum occupied by the hernial swelling. Macready attributes the origin of this form of hernia to a "wasting or congenital defect" of the muscles below the level of the anterior superior spine.

B. Direct Inguinal Hernia

Direct Inguinal Hernia is always acquired. It is in reality a ventral hernia occurring at the semilunar line. The conjoined tendon, or the tendon of the transversalis, may be split, or, more usually, carried in front of the hernial mass. Direct hernia is the hernia of middle age. A hernia appearing for the first time in a man of fifty years of age or over is probably of the direct variety. Among 63 single direct hernia 14 were scrotal and 49 in the canal; and among 61 double direct, 36 were bubonoceles, 5 were scrotal, and in 20 the older hernia was scrotal, and the recent one bubonocele (Macready).

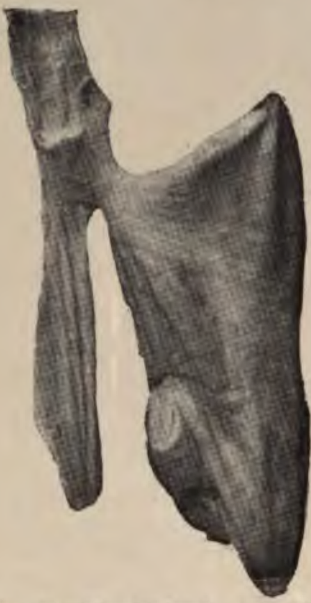


Fig. 11.—Bilocular hernia. "Two thin membranous sacs, communicating with each other and having a common opening at what appears to be the internal abdominal ring. The sac contained a hernia and a quart of 'ascitic' effusion." The identity of this condition with bilocular hydrocele is at once apparent. (Guy's Hospital Museum, No. 1159.)

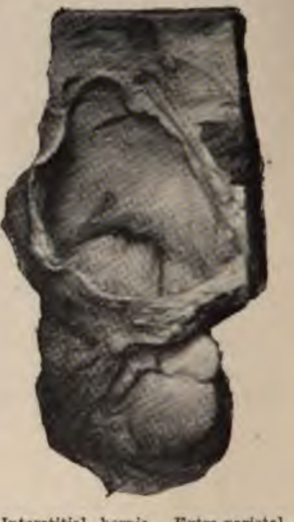


Fig. 12.—Interstitial hernia. Extra-parietal form. The sac is hour-glass in shape, the constriction being situated at the external ring close below which in the lower sac is seen the undescended testis. The upper sac extends upon the anterior abdominal wall for a distance of two inches above the widely-dilated internal abdominal ring. "T. S., aged 36 years, was admitted under Mr. Cock with symptoms of strangulated hernia of 24 hours' duration. He was found to have one tumour in the scrotum and another in the groin. The scrotal tumour was reduced by taxis. On the following day the patient died and at the autopsy nearly a foot of ileum was found strangled in the upper sac." (Guy's Hospital Museum, No. 1120.)

Signs and symptoms of Inguinal Hernia.—The general signs and symptoms of reducible and irreducible herniæ have been already set forth, and it only remains to emphasise the special features occurring in examples of inguinal rupture. In the earliest stage a mere bulging at the internal ring may be observed during expulsive efforts. By degrees the canal fills up until the external ring is reached, when the hernial swelling tends to increase rather more swiftly and to become globular in form. In a direct hernia there is no obliquity of the neck, but the mass projects directly out-

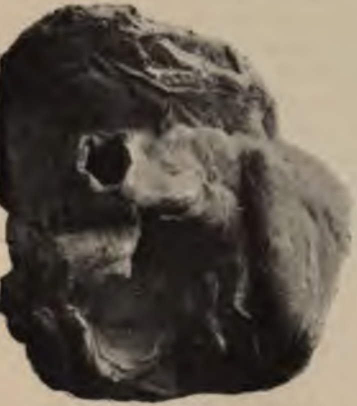


FIG. 13.—Properitoneal hernia. Ante-vesical form of diverticulum. The orifice is the ostium abdominale; one sac passes down the inguinal canal, and the diverticulum, seen opened, extended "downwards into the pelvis, by the side of the urinary bladder to which it was attached." (St. Bartholomew's Hospital Museum, No. 2118.)

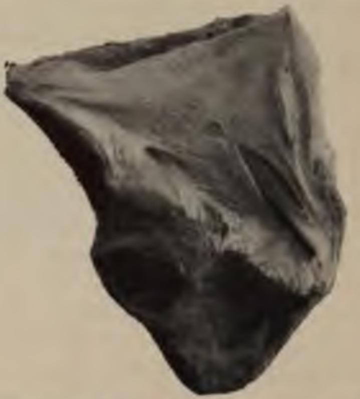


FIG. 14.—The inguinal region of a patient who suffered from right interstitial hernia. The rod leads to a large diverticulum from the sac, lying under the external oblique. The sac can be seen in the upper cut edge of the specimen. (St. Bartholomew's Hospital Museum, No. 2157A.)

wards. On examining with the index finger invaginated through the external abdominal ring there is a striking difference in the two forms of rupture. In oblique hernia the finger passes upwards and outwards along the canal and perceives the arching fibres of the internal oblique and transversalis. In the direct form the finger passes at once backwards into the abdomen. A congenital hernia may at its first appearance traverse the whole length of the processus vaginalis and reach the testis. A hernia into the funicular process may immediately descend well into the scrotum; if so, it will be cylindrical rather than globular in form. An infantile hernia has no special clinical features.

A properitoneal hernia is generally first recognised when symptoms of acute or subacute strangulation develop. In those cases where the diverticular sac projects outwards or backwards a swelling may be noticed. In 22 cases out of 36 recorded by Breiter, a "tumour or tumefaction" was noticed in 22. This proportion is probably in excess of the truth, for many cases of properitoneal hernia are recognised only as examples of "reduction in mass."

In interstitial hernia the mural sac is always capable of being recognised. At one time or another the sac can be seen distended with the usual hernial contents, and no difficulty will be experienced in the recognition. The symptoms are inconspicuous, and strangulation is infrequent.

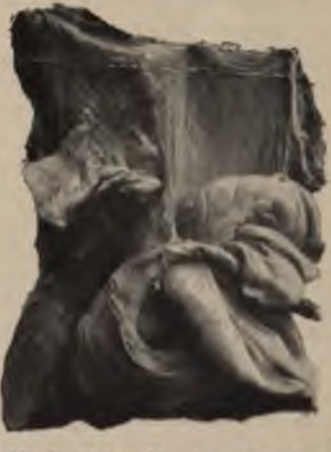


FIG. 15.—Properitoneal hernia seen from behind. Backward and outward diverticulum of the sac. An inguinal hernia was strangulated; operation was performed, and the hernia was found to be congenital. Reduction was effected. After death it was seen that reduction had occurred into a sac, shown in the figure, below the internal abdominal ring, outside the peritoneum. The specimen is labelled "Reduction in mass." The testis is retained. (St. Bartholomew's Hospital Museum, No. 2117.)

Differential Diagnosis.—From *Femoral Hernia.*—The difficulty in distinguishing inguinal from femoral hernia is far greater in women, especially stout women with indefinite landmarks and small ruptures, than in men. The crease in the skin of the groin is at its inner end, very close to the pubic spine. If this point be found and the finger placed upon it, the hernia above and internal to the finger is inguinal, and below and external to it femoral. If the hernia has reached the labium or the scrotum it must, of course, be inguinal. If the hernia be reducible, the point of its emergence from the internal abdominal or femoral ring may be ascertained.

From *Scrotal Swellings.*—The chief faults in the diagnosis of inguinal hernia are concerned with swellings of the scrotum, hydrocele in its various forms, enlargements of the testis, varicocele, hæmatocele and hydrocele of the cord. In examining a tumour occupying the scrotum it is necessary, in the first place, to determine whether the tumour is primarily scrotal or primarily abdominal, and an attempt must therefore be made to define the upper end of the swelling. In all primary scrotal swellings except infantile hydrocele, the upper limit of the tumour can be determined and the cord felt there. In infantile hydrocele the processus vaginalis is closed only at the internal abdominal ring, and fluid accumulates below this point.

A *vaginal hydrocele* is a scrotal tumour, generally rounded in outline, fluctuating and translucent. There is no impulse on coughing. If the early history can be obtained, it will be found that the swelling began at the testis and not at the inguinal canal.

A *congenital hydrocele* is translucent, fluctuating, and reducible. The reduction is slow and even, there is neither slip nor gurgle. The tumour returns gradually, and the bottom of the scrotum is first occupied.

A *hydrocele of a hernial sac* has been already described. It presents the same features as a vaginal hydrocele, but may have a thick, solid neck.

A *hydrocele of the cord* is rounded and translucent, and feels like a grape slipping along the cord. It may be reducible, and descends upon coughing. Traction on the testis generally communicates a slight movement to the hydrocele.

A *varicocele* is reducible, reappears on standing, and may have a decided impulse on coughing. The impulse, however, gives no shock to the hand, but is merely the result of the turgid condition of the veins. Reappearance of the swelling is prevented only by forcible pressure along the canal when the patient is standing; a gentle pressure sufficient to retain the bowel or omentum permits the reappearance. The irregular knotted "worm-like" feel of the swelling may simulate omentum fairly closely.

The solid tumours of the testis present little difficulty in diagnosis, and need not be further considered.

From *labial swellings.*—A hernia of the labium is practically always reducible. Cysts of the labium and hydroceles of the canal of Nuck are irreducible, smooth, translucent, and fluctuating. Varices of the labial veins may, when large, resemble hernia, but a dilatation of the surface veins will generally suggest the correct diagnosis.

INGUINAL HERNIA IN CHILDREN

Inguinal hernia is very commonly seen in children of both sexes, forming in males 23·4 per cent, and in females 22·5 per cent of all cases. It is more frequent upon the right side, and descends almost invariably in a partially or completely unclosed vaginal process. The diagnosis of the condition is rarely a matter of doubt or difficulty.

The most active of the determining *causes* is ill-feeding, leading to gaseous distension of the intestine and increased intra-abdominal pressure; others of less influence are phimosis with adhesion of the prepuce to the glans and retention of the secretion of Tyson's glands, vesical calculus, rectal polypus, intestinal irritation by parasites, or any condition leading to persistent straining on the part of the patient.

Treatment.—The very great majority of patients can be satisfactorily treated by trusses. The "wool-truss" has been much vaunted as an efficient, simple, inexpensive instrument. Personally I have been most dissatisfied with it, and now have abandoned it entirely. The best form of truss is a "spring truss," covered with india-rubber of the best quality. Attention must be paid to the proper fitting and adjustment of the truss, and to cleanliness and dryness of the skin. The truss may only be removed for purposes of cleanliness. As to the length of time for which the truss must be worn, Mr. Langton, whose experience is unrivalled, gives the following instructions:—"When the protrusion takes place before the age of 1, the use of the truss should not be discarded under any circumstances till the age of 4 years; if a truss has not been worn till the age of 3 or 4, it must be worn till the age of 10; if not worn till the age of 7, then the truss should be worn till puberty."

Operation is rarely called for. It should only be advised—

1. In cases of irreducible omentum.
2. In all cases where fluid is present in the sac.
3. In operations for the relief of strangulated hernia.
4. In all cases where it is impossible to return and control the hernia by mechanical appliances.
5. In cases where a truss has been worn for 3 or 4 years without benefit.

Strangulated Hernia in Children.—Strangulated hernia requiring operation during infancy is a very unusual occurrence. The most recent contributions to the literature of the subject have been published by Carl Stern and by Tariel of Paris. The extreme rarity of the condition is appreciated by both writers. Stern, in order to determine its frequency, consulted the records of the children's hospitals of Basle, Prague, Breslau, Vienna, Krakow, Frankfort, Amsterdam, Berne, and Gottingen.

In these hospitals for four consecutive years 139,000 children were treated, but there is no record of any case of herniotomy for strangulation. Of 1900 cases operated upon for strangulated hernia in various hospitals, 13 occurred in children. The proportion of cases in adults as compared with children is calculated by Stern to be in the ratio approximately of 108 to 1. Tariel, after remarking that several noted surgeons, among whom are Holmes, Gosselin, St. Germain, and Lannelongue, have never met with acute cases requiring herniotomy, tabulates the records that he has been able to collect. They number in all 128. Konig states that throughout his long surgical career he has only met with two cases requiring operation in the "early years" of childhood. Nussbaum operated upon two cases among a total number of 54,000 children under his care. Broca found strangulation in 9 cases out of 200 requiring radical cure in infancy. The first collection of records of operations of this kind was made by Ravoth, who tabulated 30 well-authenticated cases. Fére, who followed him, investigated the records of 52 operations in 56 cases. Howard Marsh in 1874 collected 47 cases. Knobloch, whose investigations preceded those of Stern, compiled notes of 87 herniotomies for inguinal, 11 for umbilical, and 1 for femoral hernia. To this already comprehensive list Stern added 51 later cases of inguinal and 3 of umbilical hernia.

It is remarkable that operations are more frequently called for during the earlier months of childhood than during the later. The greatest number occur in the first, second, and third months of the first year. The proportion after this period diminishes very rapidly, as is shown by comparing in a tabular form the cases operated upon in each of the first twelve months.

During the	1st	month	16	cases occurred.
”	2nd	”	15	”
”	3rd	”	14	”
”	4th	”	9	”
”	5th	”	4	”
”	6th	”	7	”
”	7th	”	3	”
”	8th	”	6	”
”	9th	”	9	”
”	10th	”	3	”
”	11th	”	3	”
”	12th	”	4	”

The symptoms and the signs are similar to those already described as being present in adults. In nearly all the recorded cases retention of urine has been observed.

Treatment.—If careful taxis prove unavailing, resort will be had to operation, which is carried out as in adults. In young children a "radical cure" is generally effected by the simple removal of the sac.

Sudden death after operation without evident or sufficient cause has been noticed by several surgeons.

FEMORAL HERNIA

Femoral or Crural Hernia (merocele) escapes from the abdomen at the crural ring, passes down the crural canal and emerges upon the thigh through the saphenous opening. It is only very rarely congenital in origin.

The crural ring is marked on the abdominal aspect by a slight depression, the *fovea femoralis*, and is closed by a specialised portion of the subperitoneal tissue, containing a lymphatic gland, known as the "septum crurale" of Cloquet. The ring is bounded *in front* by the superficial and deep crural arches, *behind* by the pubic bone covered by the pectineus and the pectineal fascia, to the *outer side* by the inner partition of the crural sheath which separates the ring from the external iliac vein, to the inner side by Gimbernat's ligament and the other fibrous structures attached to the ilio-pectineal line.

The epigastric artery is placed at the upper and outer portion of the ring, and the pubic branch of this vessel runs transversely inwards to Gimbernat's ligament. In about 30 per cent of cases the obturator artery arises from the epigastric and may then pass down on the outer side of the ring (54 per cent), directly across the ring (37 per cent), or on the front and inner side of the ring (9 per cent).

The upper part of the pectineal fascia is considerably strengthened by a tough white band of dense fibrous tissue running along the ilio-pectineal line; some of these fibres are continued into Gimbernat's ligament, and others pass across the middle line and meet those of the opposite side. This band is known as *Cooper's ligament*, or the ligament of the pubis.

The crural canal is about half an inch in length. In front of it the upper margin of the saphenous opening arches inwards to join Poupart's and Gimbernat's ligaments. That portion of the upper edge lying internal to the femoral vein is known as Hey's ligament or the femoral ligament.

A hernia passing along the canal descends at first vertically, but on reaching the saphenous opening it bulges forwards, and afterwards tends to enlarge in an upward and outward direction.

The coverings of a femoral hernia are skin, superficial fascia, cribriform fascia, the femoral sheath, the septum crurale and subperitoneal tissue, and the peritoneum. The femoral sheath and the septum crurale are generally blended into one firm layer described by Cooper as the "fascia propria."

Femoral hernia is more common in the female than the male, probably because the pelvis is proportionately wider and the crural ring actually larger than in the male. The symptoms of the rupture are similar to those present in cases of inguinal hernia, but are of a distinctly less aggressive type. A femoral hernia may pass quite unnoticed, especially in stout women, and indeed the abdomen may be opened upon a diagnosis of acute obstruction when the symptoms are due to a strangulated femoral partial enterocele of inconspicuous size.

Differential Diagnosis.—The diagnosis of femoral from inguinal hernia has already been discussed. The other conditions simulating femoral hernia are—

Adenitis.—A single enlarged femoral gland placed over the saphenous opening may very closely simulate an irreducible epiplocele. Such a gland is superficial, movable from side to side, and has no deep attachment; in all these points differing from a rupture.

Varix of the Saphena Vein.—A varix occupies almost exactly the position of a femoral hernia, has an indistinct impulse on coughing, increases in size upon standing, and is reducible. The venous distension will, however, reappear on standing if pressure be kept up on the crural canal. As a general rule, there are other dilatations of the veins of the leg and thigh on the same side.

Hydrocele of a hernial sac.—This may very closely simulate a hernia, but the fluctuation and possible translucence are distinguishing features. A distension of the *ilio-psoas bursa* may form a swelling resembling very closely in contour and feel, but differing in position from, a hernial protrusion.

Psoas abscess appears outside the femoral vessels. There is an impulse on coughing and fluctuation between the iliac and femoral portions can generally be elicited.

Hypertrophy of the subperitoneal fat may result in the presence of a tumour in the crural canal and at the saphenous opening; the down growth of this mass may drag into the canal a small protrusion of peritoneum. The condition is physically identical with an irreducible femoral hernia, but the history generally shows that the tumour has been very slow in growth and, from the earliest beginnings, irreducible.

The following rare varieties of femoral hernia have been described:—

1. *Cloquet's Hernia* (pectineal hernia), where the hernia lies beneath the pectineal fascia, on the pectineus muscle.

2. *Laugier's Hernia,* where the sac is protruded through an aperture in Gimbernat's ligament.

3. *Hesselbach's Hernia,* where a series of diverticula are sent off from the sac through the openings in the cribriform fascia. This form was described in 1814 by F. C. Hesselbach.

4. *Cooper's Hernia,* where a series of diverticula are sent off from the sac through openings in the superficial fascia.

5. *External Femoral Hernia*, where the sac descends between the femoral artery and the anterior superior spine. This form was first described by A. K. Hesselbach in 1829, but is often incorrectly described as Partridge's hernia.

Cruro-properitoneal Hernia is decidedly rare. The additional sac passes inwards towards the pelvis. Cases of "reduction in mass" have been recorded by Farabœuf and others.

UMBILICAL HERNIA

Umbilical Hernia is of three varieties—

1. Congenital Hernia.
2. The Hernia of Infants.
3. The Hernia of Adults.

1. *Congenital Hernia.*—At the beginning of the third month of intra-uterine life the viscera become enclosed in the abdominal cavity by the growth of the visceral plates. If this growth is defective, the visceral enclosure is more or less incomplete and a "hernia" results. The term "hernia," as Malgaigne suggests, is inappropriate, "for we are not concerned with viscera escaped from a cavity, but with viscera which have never entered it."

Three classes of this hernia are described—

(*a*) Where the gap at the umbilicus is small; hernia of the root of the cord.

(*b*) Where the gap is moderate in size; sacculated hernia.

(*c*) Where the abdominal wall is grossly incomplete (eventration); the viscera having a thin covering derived from the amnion.

In group (*a*) the pressure of a pad and bandage will generally effect a cure. In group (*b*) operation is necessary. The cases in group (*c*) are of merely pathological interest.

2. *The Umbilical Hernia of Infants.*—This form arises after closure of the abdominal wall, and is due to a yielding of the umbilical cicatrix. It is very frequent in both sexes, appears some weeks or months after birth, and is probably due to intestinal disorder. Almost without exception the cases are spontaneously cured. It is only necessary to apply a large firm pad and bandage to prevent protrusion of the hernia when the infant cries or strains.

3. *The Umbilical Hernia of Adults.*—This form of hernia develops comparatively late in life, and is not concerned with the variety just described. It is exceedingly rare for an infantile umbilical hernia to remain until adult age.

If the anterior abdominal wall be examined from behind, and the peritoneum be removed, a local thickening of the transversalis fascia in the neighbourhood of the umbilicus will be observed. Interlacing bundles of horizontal fibres form here a distinct layer known as the "fascia umbilicalis" or the "fascia of Richet." The extent of this fascia and its strength vary within considerable limits. Sachs, who has investigated this matter with conspicuous ability, describes three varieties as occurring:—

(1) A dense fascia most strongly developed behind the umbilicus, and thinning off above and below.

(2) A fascia of similar character so far as the upper portion is concerned, but with a sharply-defined concave margin below. The level of this margin may be lower than, or opposite to, the umbilicus.

(3) A thinner fascia situated entirely above the umbilicus.

Between the fascia of Richet and the linea alba is a passage, the "*canal of Richet,*" which contains fat, the ligamentum teres and four or five small para-umbilical veins. The entrance to the canal is directed upwards.

In the second and third varieties of the fascia it is occasionally noticed that small peritoneal diverticula are present at the lower sharply-defined concave margin. These pouches may be quite inconspicuous, or they may pass outwards through the umbilicus. Malgaigne and others have looked upon these diverticula as congenital in origin, but according to Sachs, whose observations I have verified, they are never seen before the second month of extra-uterine life. When present they may be considered as predisposing to the onset of hernia.

The lower portion of the umbilical scar has been shown by Herzog to be denser and stronger than the upper. The umbilical arteries are said to be enveloped in a firmer and more fibrous sheathing than the vein, and when the cicatricial obliterative process ensues, the scar formed around them is correspondingly more resistant and tougher than that around the thin-walled vein.

Kocher has described two forms of hernia occurring at the umbilicus in adult life: an *oblique*, which descends along the canal of Richet, lies between the fascia of Richet and the linea alba, and escapes at the navel; and a *direct*, which passes immediately outwards at the level of the umbilicus. So far as I am aware, the only author who has verified Kocher's description of the oblique form is Jaboulay, and that only in a single case. It was taught by Petit, Scarpa, and many other surgeons, that an umbilical hernia did not escape *at* the umbilicus, but through an adjacent opening, either above or below, but more commonly above. It is not improbable that some of the examples of so-called "umbilical" hernia are in reality cases of ventral hernia through the linea alba (adombilical).

Signs and Symptoms.—This form of rupture is usually the prerogative of the corpulent. Women, especially those who have borne children, are affected far more frequently than men.

In 775 cases collected by Macready, 566 occurred in women, and 209 in men.

The rupture appears very gradually, and may for a time pass unnoticed; it forms at first a smooth, globular, painless swelling, With increasing size it becomes irregular in shape; sulci appear and divide the mass into lobules of varied size. Such depressions are due to adhesions of the contents to the sac wall. The coverings of the hernia are always thin, but increasing distension from within and the chafing of clothes upon the surface may cause them to ulcerate and very occasionally to give way. The swelling, when large, is pendulous and pedunculated. The umbilical scar, stretched and considerably altered in appearance, is seen usually upon the lower half of the swelling. A rupture of even moderate duration is almost without exception irreducible. The omentum is always a content of the sac and generally lies anteriorly; this fact accounts for the lobulated irregular feel that most herniæ possess, and for the frequency of the irreducibility. The *symptoms* of an umbilical hernia are, as Sir Astley Cooper first said, more pronounced than in any other common variety of rupture. Pain, flatulence, vomiting and intestinal irregularity are certainly more frequent, and tenderness of the hernia is decidedly more pronounced. Incarceration is oftener seen than in any other rupture. Strangulation does occur, but not so frequently as is generally supposed; when present, it is generally the outcome of, and the final event in, a somewhat prolonged obstruction.

Treatment.—In the early stages, when the hernia is small and perhaps reducible, the best support is obtained from a broad, sufficiently strong, and well-made abdominal belt with a large round shield to cover the umbilical area. If the hernia is of old standing, large and irreducible, a truss with a pad of chamois leather will be needed. The circumference of the bag must consist of an oval plate, and the bag should be made of a depth rather less than that of the hernial bulging, so that a very slight degree of pressure may constantly be exerted upon the rupture. In addition, careful attention must be paid to the diet, and the patient, if inclined to obesity, should endeavour to effect a reduction in the body weight.

But an umbilical hernia, however rigidly cared for, is a constant source of danger, and therefore, whenever possible, and whenever safe, an operation should be advised. The general condition of those who suffer from this form of hernia, however, is not as a rule very satisfactory. They are persons of indolent habit, loose-bodied and flabby, and they do not readily brook surgical intervention. If, however, there is no definite hindrance, it is probable that there is more security in operation than in the treatment by trusses.

Umbilical herniæ are especially apt to become incarcerated, and the mortality of operation in times of stress is, as we have seen, considerable. The cases for operation must of course be selected with considerable care, but when so selected there is no reason to suppose that the mortality is unusually large.

Operation.—If the swelling be large, two incisions enclosing an ellipse of skin will be made, and the superfluous skin removed. On opening the sac the bowel will be returned and the omentum removed. In order to effect a satisfactory and permanent closure of the opening, it is advisable to stitch the abdominal layers up separately.

This procedure I generally carry out in the following manner. The peritoneum is first stitched up with a continuous layer of catgut. The inner rounded margin of the rectus sheath on each side is then divided for the whole length of the wound by a vertical incision midway between the anterior and posterior surfaces, until the muscular fibres are reached. Then the posterior part of the sheath is stitched to its fellow across the middle line by a series of interrupted silkworm-gut sutures. Similarly the muscle-fibres of the two recti are stitched, and afterwards the anterior layers of the sheaths. Finally, the skin and subcutaneous tissues are closed in the usual manner. There are thus five layers of sutures, and four of the layers are buried. For the buried stitches I prefer silkworm-gut, which can be effectively sterilised, but kangaroo tendon or strong chromicised catgut answers very well.

There can be no doubt that recurrence after operation is more frequent in umbilical hernia than in inguinal or femoral; and for this reason it is always a wise precaution to insist upon the subsequent wearing of a stoutly-built abdominal belt.

Ventral Hernia

The term *ventral hernia* is held to include all such ruptures of the abdominal parietes as do not appear at the inguinal, femoral, or umbilical apertures. Any portion of the wall may be the site of such a protrusion. For purposes of description the following varieties are recognised:—

1. Divarication of the recti.
2. Ventral hernia in the linea alba.

3. Ventral hernia in the linea semilunaris.
4. Ventral hernia following traumatism.
5. Lumbar hernia.

(1) *Divarication or Diastasis of the Recti.*—In children it is not infrequent to notice a widening and a thinning of the linea alba above the umbilicus. On crying or straining a semi-cylindrical protrusion occurs, and extends from the ensiform cartilage to the umbilicus. Clinically the condition is unimportant, and treatment is not necessary. In adults, and especially in multipara, the separation of the recti occurs below the umbilicus. The fingers can be readily passed into the gap. Rossetus relates a case occurring in a pregnant woman, in which the fœtus could be plainly felt in the hernial mass.

(2) *Ventral hernia in the linea alba* may occur above or below the umbilicus. The former is generally known as *epigastric* hernia, and occurs more frequently in men than in women. The linea alba above the umbilicus is about 3 mm. in width and consists of transverse fibres. It is no uncommon experience to find that these fibres are decidedly coarse in texture and small irregular apertures or spaces are left between them. Through these a protrusion of subperitoneal fat occurs, forming a rounded nodule readily felt on palpation. Such a mass may increase considerably in size and eventually drag outwards a peritoneal pouch, which may form a hernial sac, containing omentum, intestine, or both. Very rarely the stomach has been recognised as a hernial content.

These ruptures are chiefly remarkable for the persistent symptoms they induce. Dyspepsia, sickness, colic, and general intestinal discomfort are frequently complained of, and in not a few cases their cause may be overlooked or ignored. The treatment by operation should, if possible, be the routine procedure.

Below the umbilicus a ventral hernia, apart from those varieties to be mentioned, is very exceptional.

(3) *Ventral hernia in the Linea Semilunaris.*—A direct inguinal hernia is in reality a hernia at the linea spigelii. Apart from this form examples are rare. The majority of recorded cases have occurred below the umbilicus, and Mollière (whose name is attached to this rupture by French writers) suggested that the fold of Douglas determines the point of exit of the protrusion.

A very curious feature of herniæ in this situation has been described by Monro, Teale, and others. The hernial sac may enter the abdominal wall without passing through it, and consequently there is no manifest swelling on the body surface. The term "Masked Hernia" has been applied to this condition.

(4) *Ventral hernia following traumatism* may be found at any portion of the abdominal wall. Guthrie describes many cases occurring during the Peninsular War as the result of blows inflicted on the abdominal parietes. Any of the numerous operations practised upon the abdominal contents may leave a permanent weakness in the scar, which gradually yields under pressure from within. Cicatricial tissue is not suited to withstand pressure. Such herniæ can be almost entirely obviated by careful suturing, layer by layer, of the divided structures. Wherever possible, muscle-fibres should be separated in the direction of their length rather than cut. Mr. Howse first introduced this most important principle into surgical methods in performing gastrostomy, and MacBurney and others have carried out the same idea in operating upon the appendix. In every abdominal operation much can be done in this way to preserve intact the muscular structure.

The best treatment in the majority of cases consists in the use of appropriate specially-designed belts. An operation for the cure of such hernia is not attended with a large measure of success, and should only be employed in very favourable cases.

LUMBAR HERNIA

This form of hernia is rare, less than fifty examples being recorded. In more than half of these no mention is made of the precise point of exit.

In the lumbar region are two triangles, the triangle of Petit and the triangle of Grünfeldt, or the "upper" and "lower" lumbar triangles. The former is bounded by the anterior edge of the latissimus dorsi, the posterior edge of the external oblique, and, at the base, by the iliac crest. It is present in about 25 per cent of children and 75 per cent of adults. The latter is bound by the twelfth rib and the serratus posticus inferior above, the internal oblique below and in front, and the outer edge of the quadratus lumborum below and behind. The floor of the triangle of Petit is formed by a stout fascia covering the internal oblique, and beneath this by the aponeurosis of the transversalis. The upper triangle is roofed in by the latissimus dorsi, and in the floor lies the lumbar aponeurosis alone; it is probably the weakest area in the lumbar region.

The triangle of Petit is the most usual point of exit of a rupture, being so mentioned in eleven cases; in three cases certainly, possibly in four, the hernia has passed through Grünfeldt's triangle. In the remaining cases the distribution has been quite irregular. As causes of the hernia may be mentioned traumatism, abscess formation, congenital defect of the muscles, and the presence of lipomatous masses derived from the subperitoneal tissue, which in their outward bulging drag small peritoneal pouches with them.

The hernia is rather more frequent in men than in women, and on the left side rather than on the right. The symptoms and signs are those of an ordinary hernia. The swelling is soft and globular and generally reducible. In a few cases strangulation has necessitated operation. It has been observed on dissection that the sac has returned with the contents when taxis was applied. The conditions for which hernia may be mistaken are abscess, hernia of muscle, sarcoma, lipoma, and hæmatoma.

Treatment can be carried out by means of an abdominal belt, by the wearing of a specially made truss, taking its fixed point from the opposite hip or by operation. The latter has been practised by Owen, Coze, Giordano, and Zucker.

OBTURATOR HERNIA

An obturator hernia escapes from the pelvis at the opening for the obturator vessels and nerves. On the under surface of the horizontal ramus of the pubes is a groove which passes from behind, downwards, forwards and inwards, and according to Vinson has a length of 2 cm. Along this groove the bowel or other viscus passes, carrying before it the peritoneum, the subperitoneal tissue and the pelvic fascia. In its onward course the hernia may pass between the obturator membrane and the obturator externus, above the obturator externus, or beneath that upper slip which is occasionally separated from the body of the muscle by the passage of one or both divisions of the obturator nerve. The obturator nerve is generally found on the outer side of the sac and the artery behind, or behind and to the outer side. The most usual content of the sac is the small intestine; but the ovary, Fallopian tube, uterus, and bladder have all been found therein.

Symptoms and Signs.—The majority of the recorded cases have been recognised only after the onset of symptoms of acute intestinal obstruction. When a tumour is present it is most readily felt from the inner side of the thigh behind the adductor longus, when the limb is flexed, rotated outwards and adducted. A fulness in Scarpa's triangle may readily escape detection, especially in women, in whom this variety of hernia is very much more frequent than in men. In some cases the passage of intestine through the canal may be felt on rectal or vaginal examination. In one patient, examined by Macready, the finger, when made to sweep round the margin of the obturator foramen, was stopped by a firm cord, about the thickness of the thumb, which could be felt to enter the obturator canal. Pain may be elicited by making tense the obturator externus muscle. Disorders of sensation along the course of the obturator nerve are met with in about half the cases. There may be pain or numbness along the inner side of the thigh or in the groin, the hip, or the leg. This symptom of obturator neuralgia, generally called the "*Howship-Romberg*" *symptom*, even when present, has been frequently misunderstood, and may indeed be produced by other conditions than obturator hernia.

Treatment.—The mortality of strangulated obturator hernia is 85 per cent. If a hernia were diagnosed it would be almost impossible to adapt an effective truss. There is, therefore, every reason to urge that in all cases operative measures should be practised. The sac can be exposed by an incision parallel to the femoral vein and about 1 inch internal to it. The interval between the pectineus and the adductor longus is sought and the muscles separated or the former divided. Hæmorrhage, copious in quantity and difficult to control, has resulted from blindly incising the obturator membrane. In order to avoid this the structures should be exposed, and this procedure will be facilitated by the adoption of the Trendelenburg position and the abduction of the thigh on the affected side. In certain cases the reduction of the hernia through a median abdominal incision may be advisable.

Other varieties of hernia, such as ischiatic, pudic, pudendal, etc., are so rare that no description of them is required in this place.

HERNIA OF SPECIAL VISCERA

There are some forms of hernia which derive their chief importance and certain of their intrinsic signs and symptoms from the inclusion of special viscera, such as the bladder, the ovary, and the vermiform appendix. These require, therefore, individual mention.

HERNIA OF THE BLADDER.—The frequency of bladder implication has been very differently estimated by various observers. As a rule it may be assumed that those operations wherein an opening up of the inguinal canal is practised are the most likely to reveal the bladder descent.

In 2543 operations for hernia tabulated by me in my "Arris and Gale" lectures for 1900, there were twenty-three cases of bladder hernia, giving approximately 1 per cent. Broadly speaking, it may be affirmed that this hernia is a disease of the aged and enfeebled. Only five cases are recorded as having occurred in children.

A hernia of the bladder may be present in the inguinal, femoral, perineal, obturator, and ventral forms of hernia. The inguinal is the most frequent, forming more than 90 per cent of the recorded examples.

Varieties of Cystocele.—Three varieties of cystocele are recognised:—

1. *Intra-peritoneal.*—An ordinary oblique inguinal hernia is present;

into the sac a portion of the bladder, *completely covered by peritoneum*, descends, the portion of the viscus implicated being the upper part of the posterior surface.

2. *Para-peritoneal.*—The inguinal hernia may be oblique or direct. On the inner side of the sac lies the bladder, in such manner that the peritoneum of the inner wall of the sac is the serous covering of the outer wall of the bladder. The rest of the bladder outside the abdomen has no peritoneal coating. The viscus, therefore, is not a content of the sac, but projects into it. This form is the most frequent.

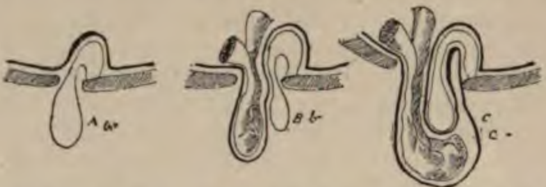


FIG. 16.—The three varieties of cystocele. A. Extra-peritoneal; B. Para-peritoneal; C. Intra-peritoneal.

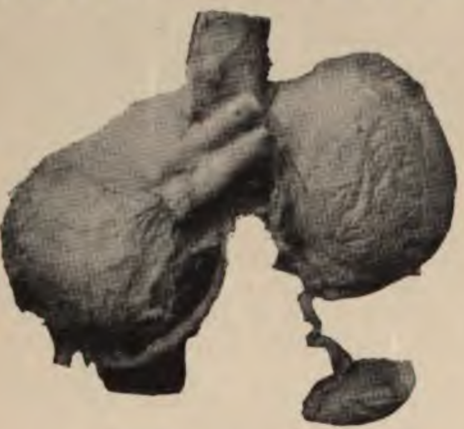


FIG. 17.—Vesical hernia. Sir Astley Cooper's case. The bladder is covered by peritoneum except at the inner and posterior part. Para-peritoneal form. (Guy's Hospital Museum, No. 1170.)

3. *Extra-peritoneal Cystocele.*—This form is rare. The bladder uncovered by peritoneum escapes from the abdomen on the inner side of the deep epigastric artery; the hernia, that is to say, is of the direct form.

The bladder may generally be recognised by the copious deposit of fat which surrounds it. In a very large number of cases the bladder has been wounded, being mistaken for the sac, a second sac, a cyst, or a lipoma.

Symptoms and Signs.—Very few cases of hernia of the bladder have been diagnosed before operation. When, however, a large portion of the bladder is implicated (the whole bladder, including the prostate, has been twice herniated) the symptoms are unmistakable. In such a case the signs are those of a hernia which at times contains fluid. The tumour is rounded, smooth, soft, fluctuating; the fluid is capable of reduction, and a desire to urinate is then created. The size of the hernia is subject both to rapid and to considerable alteration. The functional characteristics are striking. The chief of these is the "miction en deux temps," the divided micturition. A patient so affected voids urine and empties the portion of the bladder in the pelvis. Then, on pressure being applied to the hernia, the urine it contains is returned within the pelvic portion of the bladder and thence passes by the urethra. Micturition is in two stages and is associated with a simultaneous lessening of the hernial swelling. If the bladder, after being naturally emptied, is filled by the injection of lotion, the fluid will pass through the canal and distend the hernial tumour.

Treatment.—In operating upon all herniæ, especial attention should be paid to any thickening of the inner side of the neck of the sac, and more particularly to a deposit of fat in this situation, for by such signs is the presence of the bladder indicated. If the bladder be wounded, the wound should be stitched by a double layer of sutures and may then be safely reduced, and the operation completed in the usual manner.

As a truss cannot be safely worn in cases of cystocele, an operation should be resorted to whenever possible.

The accompanying table gives, at a glance, the chief features of the recorded examples of bladder hernia.

HERNIA OF THE BLADDER.

Total recorded cases, 171.

Cystocele diagnosed; no operation					13 cases.
(In three of these, diagnosis was confirmed after death.)					
Cystocele diagnosed; operation					6 cases.
Bladder wounded (resection)			1		
Bladder not wounded			5		
Cystocele found; after wrong diagnosis					4 cases.
(3 diagnosed as abscess; 1 as sarcocele.)					
Cystocele, first discovered at operation					107 cases.
Bladder recognised and not wounded			49		
„ wounded and recognised			58		
As follows:—					
Wounded accidentally, though recognised			2	58[1]	
Unexpectedly wounded			26		
Intentionally incised			23		
Being mistaken for:—					
The sac	3	23			
A second sac	8				
Tumour	4				
Cyst	1				
Omentum	2				
Lipoma	2				
Not mentioned	3				
Incised for diagnostic purposes			5		
„ to confirm diagnosis			2		

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[1] Bladder wounded and recognised 58

The cases were dealt with as follows:—

	No. of cases.	Result.		
		Healing without Fistula.	Fistula.	Death.
Bladder simply left open	2	0	2	0
„ stitched to skin	4	0	3	1
„ drained by tube	1	0	0	1
„ ligatured	3	0	1	2
Fastened in ordinary suprapubic incision, made for the purpose	1	0	0	1
Suture of Bladder—simple	10	5	2	3
„ „ double layer	15	13	2	0
„ „ triple layer	12	6	5	1
Suture; no details	10	4	5	1
	58	28	20	10

Cystocele, first recognised after operation because of wounding of bladder		22 cases.
Wound not noticed at operation	6	
Supposed to be sac	9	
,, ,, second sac	5	
,, ,, lipoma	2	
Cystocele, found post-mortem		19 cases.

Hernia of the Ovary.—Hernia of the ovary is very much more commonly inguinal than femoral, and congenital than acquired. Most of the cases are seen in infants, and the condition may then be bilateral, and be associated with malformations of the uterus, or the Fallopian tubes. In infants the condition may be only a temporary one, the ovary retiring spontaneously within the abdomen as the child grows. But at all ages irreducibility may be met with. According to Macready, 48·6 per cent are reducible in children and only 15·3 per cent in adults.

Symptoms and Signs.—In children the ovary may be felt in the inguinal canal or beyond the external abdominal ring as a small, oval, solid body, freely movable when pressed upon, and attached at its upper end by a thin cord which enters the abdomen.

In the adult the physical signs resemble those given by an adherent pellet of omentum. Occasionally movement may be transmitted to the ovary by traction upon the uterus through the vagina. If the patient menstruates (which is not always the case), the ovary will be recognised as being swollen and tender at each period.

Examples of herniated cystic ovary and parovarian cyst have been recorded. (See "Ovary.")

Treatment.—An operation is nearly always desirable. The ovary when healthy should be reduced if possible. If firmly adherent or altered by pathological processes removal will be necessary.

Congenital Hernia of the Cæcum and of the Sigmoid Flexure.—The cæcum on the right side and the sigmoid flexure on the left are occasionally present in "congenital" sacs. It has been shown that under such circumstances the visceral descent is the result of the action of the gubernaculum. Above the testis the smooth muscular fibres of the gubernaculum are continued upwards with the vessels of the testis and the vas deferens, in a peritoneal fold to which Lockwood has given the name "Plica vascularis." The ultimate attachment of these fibres is to the peritoneum lining the posterior abdominal wall. In these forms of hernia the unduly exaggerated action of the gubernaculum results in the dragging down bodily of the peritoneum to which the fibres are attached. There is, as it were, a sort of "landslip." The cæcum or the sigmoid flexure will then bear to the sac the same relation as to the parietal peritoneum before the descent began, for what was previously parietal peritoneum is now the sac, and the mutual relationship of viscus and serous membrane is unaltered.

Traces of the gubernaculum (the "natural fleshy adhesions" of Scarpa) have been discovered in the wall of the sac.

Treatment.—In the operative treatment of these cases the removal of the whole sac is, of course, out of the question. The peritoneum should be removed up to within an inch of the attachment of the viscus to the sac, the cut edges of the membrane stitched with a continuous suture of fine catgut, and the curtailed sac and its contents bodily returned.

Acquired Hernia of the Cæcum.—An acquired hernia of the cæcum is most frequently of the right inguinal variety, but right femoral, and left inguinal and femoral, are also met with. There has been much needless discussion as to the peritoneal coverings of the cæcum when involved in a hernia.

It may be emphatically stated that there is no difference in the behaviour of the cæcum in this respect from that of any other viscus. The peritoneal covering of the cæcum and ascending colon varies within very wide limits. In some examples, though very few, the cæcum may have no coating of serous membrane on its posterior surface. In others, the cæcum and the whole of the ascending colon may be included within the layers of a mesentery continuous with that of the small intestine. Between these two extremes any condition may be met with, but in the great majority of cases, at least 95 per cent, the cæcum and a small portion of the ascending colon are completely clad with peritoneum.

Whatever the condition of the cæcum may be when within the abdomen, such will its condition be when descended into a hernia. If the serous covering be absent in the abdomen, it will be absent in the hernia; if complete within the abdomen, it will be complete within the hernia.

HERNIA OF THE APPENDIX VERMIFORMIS.—Hernia of the appendix derives its chief interest from the fact that inflammation, recurring from time to time, and possibly going on to suppuration or gangrene, may occur in the hernial sac. The implication of the little process in the hernia may lead to its strangulation or to an attack of appendicitis; or, it has been suggested, the occurrence of inflammation with the attendant swelling and stiffening of the process may be primarily responsible for the hernial descent.

The appendix may be alone in the sac, or be associated with the cæcum or with other viscera. It may be normal in appearance, and reducible; it may be bulky from the deposit of fat, distended into a cyst, or shrivelled and irreducible.

So far as the treatment of the condition is concerned, it is probably safer in all cases to remove the appendix. This can generally be effected through the incision made for the treatment of the hernia, but in certain cases a second incision over the cæcum may be desirable.

THE TREATMENT OF HERNIA

The treatment of hernia is either *palliative* or *operative*.

PALLIATIVE TREATMENT consists in the wearing of mechanical appliances, specially fashioned for each separate form of rupture, known as *trusses*.

A truss should fulfil two essential conditions: it should retain the hernia completely under all circumstances, and it should be perfectly comfortable.

Inguinal and femoral trusses consist of a belt, containing a steel spring encircling the body and a pad, by means of which the force of the spring is applied to the point of exit of the rupture. Steel is better than any other metal, but is not wholly satisfactory. The secretion of the skin causes the spring to rust, and, after some time, to snap. No other material, however, is so satisfactory either in this or other respects. The pad is best made of cork covered with a layer or two of flannel. The truss throughout is covered on the external surface by leather or calf-skin, and on the inner side by chamois leather.

A patient, when measured for a truss, should be lying down. A tape is then carried round the body from the base of the sacrum behind, between the crest of the ilium and the upper border of the great trochanter laterally, and just above the symphysis pubis in front. The number of inches, in this measurement, indicates the size of the truss.

A truss should be applied by the surgeon. The leaving of this matter in the hands of instrument-makers has led to much irregular practice, and to the introduction of many "special" or "patent" trusses which are, with scarcely one exception, worthless, or positively harmful. A truss is as surely a surgical instrument as a splint, and requires at least equal care and skill in the application.

INGUINAL TRUSSES.—1. *For Bubonocele.*—A single truss should be of the exact size, obtained by measurement in the manner just mentioned; a double truss should be a size (that is, one inch) larger for its coverings are thicker and there is not the same adaptability as is afforded, in the single truss, by the strap. The inner end of the pad should be at the outer border of the rectus, and *above* (*not on*) the pubes. The truss should lie in the hollow just below the iliac crest, and should fit snugly and firmly. The commonest of all faults is to find a truss which is too large for the patient.

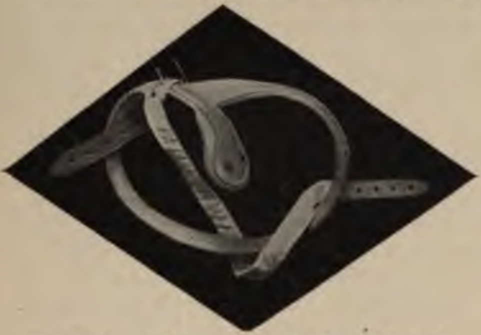

FIG. 18.—Inguinal truss to show the shape of the pad.

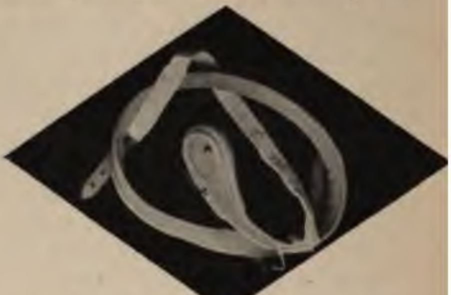

FIG. 19.—Femoral truss to show the shape of the pad and the forward attachment of the under-strap.

After placing the belt of the truss in position, the cross-strap should be first fixed, and then the under-strap, which is carried round in the fold of the buttock from the shoulder of the truss to the bottom stud on the pad.

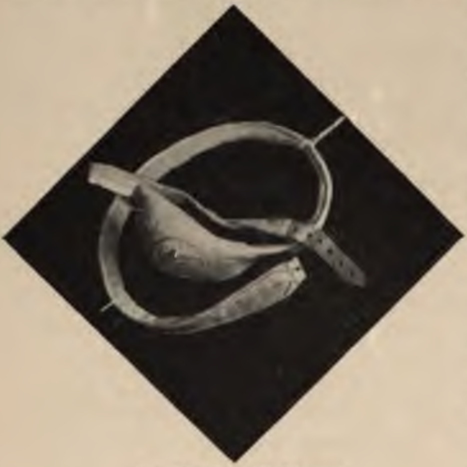

FIG. 20.—Rat-tailed truss.

2. *For Scrotal Hernia.*—The ordinary pad is not sufficient, as a rule, to retain a rupture which has descended into the scrotum; it must be made fuller and more bulky, and of such a shape that the surface next the skin looks rather more in an upward direction. The soft part of the pad should also be prolonged downwards into a tail or under-strap which can be attached to a fixed buckle or hook in front of the shoulder of the truss. The double object of exerting pressure on the inguinal canal (by the pad) and on the external ring (by the under-strap) is effected by this truss, which is known as the rat-tailed truss.

3. *For direct hernia* a rat-tailed truss or a modification known as the "forked-tongue truss" is employed. In this latter the pad is carried inwards to the middle line and terminates in the strap which is fixed to the belt of the truss in front of the shoulder of the opposite side.

Double trusses should be made the same on both sides, double rat-tailed, double forked-tongue, or double ordinary, as the need may be. A truss whose two sides are dissimilar is rarely satisfactory.

Femoral Trusses.—The same measurement is used as in the case of inguinal hernia. The pad of a femoral truss is rather smaller than that of an inguinal, and is prolonged a little downwards to cover the femoral canal. The understrap is attached to the truss well in front of the shoulder, so that when made tight it tilts the pad and causes increased pressure to be made on the femoral ring, which is normally almost horizontal when the body is erect.

The pad should be kept well outside the pubic spine and should not be allowed, as it often is, to over-ride that bony prominence. If the hernia is large and the pad has to be increased considerably in size in order to make effective pressure, a belt must be added to encircle the thigh. If an inguinal hernia is present on the same side, the femoral pad can be prolonged upwards and inwards to cover the canal and ring.

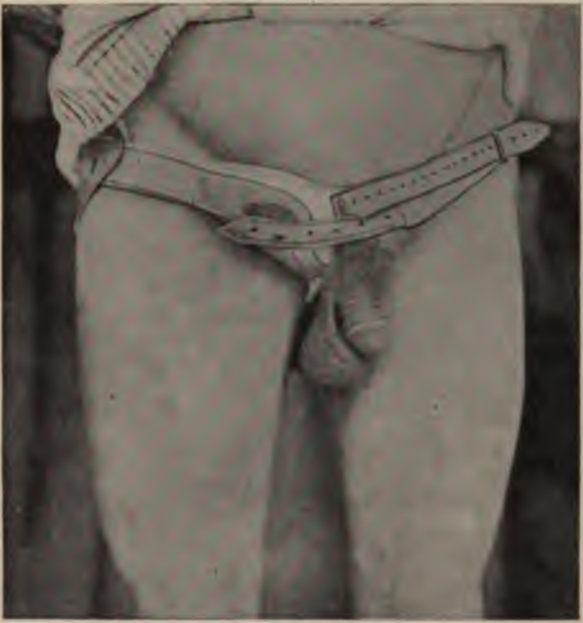

Fig. 22.—Forked-tongue truss applied.

Operative Treatment.—Since the introduction of modern methods into surgery the number of cases submitted to operation has, very properly, increased considerably. Probably, however, the question of the propriety of interference will remain for some time largely a matter of the personal choice of the surgeon. There are some surgeons who look upon most examples of hernia as legitimate cases for the "radical" cure, while others, carefully selecting their cases, have, by growth of experience, been led to believe that very few patients are really cured by operation, and they have in consequence restricted the treatment by operation to examples of congenital hernia. There are two points which should be considered before attempting any decision in the matter. The first is that, as shown by Macready and others, 36·1 per cent of cases of hernia, at first single, become, after the lapse of weeks, months, or years, bilateral. The second is that the recurrence of a hernia after radical cure is, unhappily, far more frequent than is generally conceded.

The results of Halsted and Bassini are the best so far recorded, but, allowing for the varying experience of all surgeons, one may state the likelihood of return as approximately 10 to 15 per cent of all the cases submitted to operation.

There are certain cases which, in the absence of urgent or striking reasons to the contrary, should be invariably submitted to the "radical cure." Such are—

1. Cases of uncontrollable or irreducible herniæ, where treatment by trusses is either inapplicable or has proved unavailing.
2. Cases occurring in young men, otherwise healthy and sound, desirous of entering the services.
3. Cases of strangulated hernia in which the gut has been reduced.

On the other hand, there are instances in which an operation is rigidly excluded. These are—

1. In the old, very feeble, cachectic or broken down, who are unable to tolerate any surgical treatment.
2. In young children, except in cases of strangulation.
3. In enormous herniæ, where a shrunken abdomen has resulted from the steady and progressive hernial enlargement.
4. In those cases where an inherited and extensive weakness of the abdominal muscles is the chief factor in determining the occurrence of the rupture.

Between these two groups, however, the majority of cases occur, and it is in dealing with them that the surgeon must exercise his discretion. My own feeling is that within reasonable age limits in healthy subjects, say from ten to fifty years, I should certainly advise treatment by operation in the very great majority of patients. If the patient is in good condition, if the rupture is of only moderate size, and if a complete union of the wound is obtained without suppuration, the chance of recurrence is very small indeed, probably less than 5 per cent. When, moreover, a recurrence under such circumstances does take place, the rupture is, almost without exception, easier to control by truss pressure than the ordinary complete hernia.

The Radical Cure of Hernia

1. *Inguinal.*—The treatment of inguinal hernia by operation was introduced by Celsus, who, it is interesting to note, expressly did not advocate any operative measures for ruptures which were strangulated. The removal of the testis, which for centuries was considered as a necessary incident in the operation, was advised by Paulus of Œgina.

Since the introduction of modern methods into surgical practice the operative treatment of all forms of hernia has been marked by a most florid activity. Several "essential principles" have been evolved, and the modifications of each have been bewildering in their frequency and unnecessary complexity. Probably no subject in the wide domain of medicine, not even the subject of pessaries, has had so much that is trivial and worthless written upon and around it as this.

Of the operations that have been widely used the following alone need mention:—

1. *Simple ligature of the neck of the sac* and removal (Socin).
2. Ligature of the neck of the sac and *stitching up the external ring* (Czerny, Banks, Championnière).

3. *Pleating of the sac*, which is fixed as a pad at the upper end of the canal; suture of canal (Macewen).

4. *Torsion of the sac*, with suture in the canal (Ball).

5. Torsion of the sac, *displacement of the neck*, suture of canal (Kocher). Recently modified (see description later).

6. Removal of the whole sac, *displacement of the cord*, suture of the canal (Halsted, Bassini).

There can be no question that the operation most widely practised at the present time, and incomparably the most successful in results, is that introduced by Bassini. The operation of Halsted (which has priority of publication) is the same in principle, but the details of the operation are different in certain particulars.

Bassini's Operation.—An incision is made in the inguinal region, beginning a little below and internal to the anterior superior spine, and ending at the centre of the external abdominal ring. The incision exposes the tendon of the external oblique muscle, which is split, in the direction of its fibres, from the ring upwards for about 1½ inch.

The sac is now sought for among the structures of the cord. If the hernia has never presented at the external ring this may be a matter attended with some difficulty, but a little patient clearing of the cord will usually suffice to expose the sac, which is then gently and carefully isolated up to the internal abdominal ring. The sac is now opened, the intestine or other viscus replaced within the abdomen, and the omentum, if present, removed. The sac is tied at the internal ring after transfixion and removed; or the sac is removed, and the cut edges of the parietal peritoneum at the neck stitched by interrupted or continuous sutures. The cord is then gently lifted from its bed, and held up by blunt hooks. Sutures, three, four, or five in number, are introduced through Poupart's ligament on the outer side, and on the inner through the arching fibres of the internal oblique and transversalis, the transversalis fascia, the conjoined tendon, and, if necessary, the sheath of the rectus also. The sutures are passed underneath the cord; when tied and cut short the cord is laid upon them. Finally the cut edges of the aponeurosis of the external oblique are united over the cord by a continuous suture, and the skin wound is closed. A drainage tube is not necessary. If, on laying open the canal, the veins of the cord are found to be varicose, they should be excised. At times the fat lying along the cord, continuous above with the subperitoneal fat, is greatly increased in bulk, forming a definite lipoma which must be removed. Under all circumstances the cord should be handled with care and gentleness; a rough and hasty stripping and tearing of the sac may lead to inflammatory trouble in the wound or in the testis.

If the cord is found to be so thick that the aperture left for its passage through the abdominal wall might tend to induce a fresh descent of the hernia, the cord may be split into two equal portions, the outer one being placed outside the upper stitch, and the inner between that stitch and the next. If the cord is turgid with veins which are not varicose this method of splitting is preferable to the excision of the veins, for experience has abundantly shown that the latter procedure is one not seldom attended by an attack of acute or subacute orchitis, which may, itself, be followed by a gradual withering of the testis.

When a recurrence of the hernia takes place after this operation it is generally found at one of two points, at the internal abdominal ring or at the inner and lower portion of the external abdominal ring. When at the former point it is most probably due to the bulky thickening of the cord, and may be prevented by the splitting of that structure in the manner just described. When at the latter point it is almost certainly due to the weakness of the conjoined tendon, a weakness which, as I have already mentioned, is very frequent. In order to strengthen the abdominal wall at this point the following device is useful. Before introducing any of the sutures, the sheath of the rectus muscle is opened by an incision along the outer side in the direction of the length of the fibres. The muscular bundles of the rectus are then included in the two or three innermost stitches. On tightening these stitches the muscular fibres are brought into close approximation with Poupart's ligament, and the inner end of the gap in the abdominal wall is thereby considerably strengthened. This so-called "transplantation of the rectus" was first suggested by Wöfler in 1892, but his paper has not received general recognition. Though I have practised this method for over two years I was myself unaware of Wöfler's article until I saw it quoted in Bloodgood's recent work. (See list of references.)

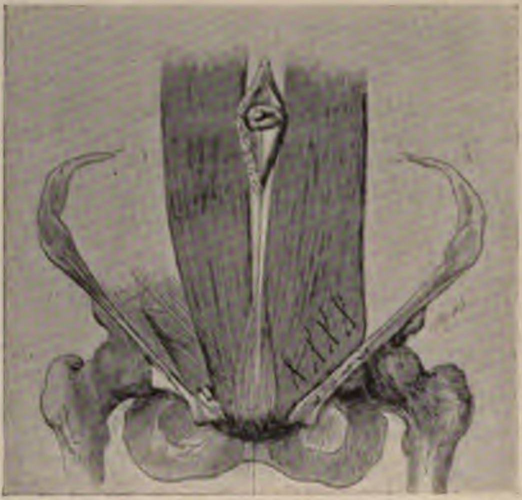

FIG. 23.—Transplantation of the rectus muscle. (Bloodgood.)

Halsted's Operation.—After exposing the arching fibres of the internal oblique, as in Bassini's operation, the free lower border of this muscle is caught with two artery clamps, placed 1 cm. apart, and the muscle cut between them for a distance of 3-4 cm., at right angles to the muscular bundles. The division of the muscle is made as far from the linea semilunaris as possible. The cord is brought out through the incision in the internal oblique and is made subcutaneous. Five Mattrass stitches, as a rule, are introduced, passing through the aponeurosis of the external oblique, the internal oblique, transversalis muscle and fascia on the

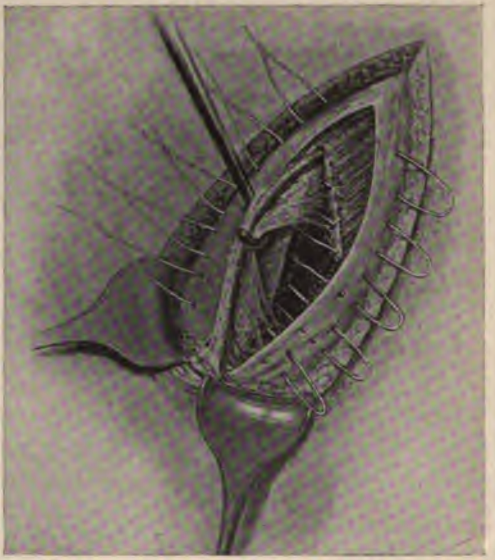

FIG. 24.—*Halsted's operation.* Showing the division of the internal oblique, the transplantation of the cord, and the method of introducing the stitches. (Bloodgood.)

one side, and through the transversalis fascia, Poupart's ligament, and fibres of the aponeurosis of the external oblique on the other.

Kocher's Operation.—Dr. Albert Kocher informs me that the operation now practised (May 1900) by Prof. Kocher is carried out as follows:—An incision is made over the inguinal canal; the sac is isolated from the cord very carefully as high up as possible, traction being maintained upon the sac in order to pull it down to its farthest extent. A special light forceps then seizes the end of the sac, which is invaginated upon itself (just as one would turn the finger of a glove inside out), and brought up, about 1 cm. above and outside the internal abdominal ring. An incision of ½ cm. is made through the external oblique aponeurosis down to the point of the forceps, which is thrust through the small wound. The sac, still inverted, is pulled out as far as possible. A silk ligature is passed round the neck of the sac and the sac removed beyond the ligature. The stump of the sac is pushed back through the opening in the fascia. The ligature surrounding the neck of the sac is left long, and with it the edges of the little wound in the aponeurosis (including the cut edges of the parietal peritoneum there) are stitched together.

Finally some six or eight sutures are passed through the anterior wall of the canal to strengthen it, and so make the outer ring smaller.

For the buried sutures many materials have been recommended. Halsted uses silver-wire and is eminently satisfied with it. Bassini, since 1892, has abandoned the use of silk, and now employs chromicised catgut. Silk, silkworm gut, kangaroo tendon and chromicised catgut are those in general use. Silk has the great disadvantage that, if the wound becomes septic the silk will be readily infected, and thereby give rise to a persisting sinus. Silkworm gut is open to the same objection, but in practice I have had very little trouble with it. Kangaroo tendon and chromicised catgut both remain unabsorbed for several weeks, and probably in that time the deep union of the wound is sound and complete. Marcy and Bull and Coley use and advise kangaroo tendon. After trying impartially all these forms of suture I now use nothing but silkworm gut.

In all operations for the radical cure of hernia a point of chiefest importance is the securing of perfect primary union. In no other operation is this so essential. Recurrence of the hernia is proportionally very much more frequent in cases that have suppurated than in those where primary union has been secured.

2. *Femoral.*—The radical cure of a femoral hernia is generally very much more satisfactory in its result than is the case with any other variety of rupture.

The operative procedure is very similar to that employed in cases of inguinal hernia, so far as the earlier steps of the operation are concerned. A vertical or transverse incision is made, the sac exposed, stripped and opened. The viscera are replaced or omentum removed, the sac ligatured as high as possible and removed. For the closure of the canal and ring two methods are in general use:—

A. *Bassini's Method.*—With a curved needle a series of sutures are introduced uniting the fascia covering the pectineus muscle with the inner end of Poupart's ligament, above and internally, and with the falciform process of Burns externally. All the sutures (five or six are generally used) are passed and are then tied from above downwards.

B. *Lockwood's Method.*—A special curved needle armed with silk is used, whose point is guided up the femoral canal until it rests against the inside of the linea ilio-pectinea, opposite the outer edge of Gimbernat's ligament. The needle is then rotated so that its point scrapes over the linea ilio-pectinea and picks up Cooper's ligament. Finally the point emerges through the upper part of the pectineal fascia, where it is unthreaded and withdrawn, leaving the suture beneath Cooper's ligament. Additional sutures are passed in exactly the same way, but each a little farther outwards, until the last lies at the inner edge of the common femoral vein. Two or three sutures generally suffice, but more may be necessary. The next step is to again thread the upper end of each ligature in turn through the herniotomy needle, and by pushing the point of the needle half-way up the femoral canal and rotating it forwards pass the thread from within outwards through Hey's ligament, close to its junction with Poupart's ligament. On tying these sutures Hey's ligament is united to Cooper's ligament, and the crural canal is firmly closed.

Lockwood's operation is most satisfactory in its results, and is, in my opinion, decidedly better than Bassini's, which closes only the lower opening of the canal, and leaves untouched the crural ring.

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